Civil Conflict, Gender-Specific Fetal Loss, and Selection: A new test of the Trivers-Willard hypothesis

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Abstract

A sizeable economics literature explores the effect of prenatal shocks on later health or socioeconomic status. Work in other disciplines, following the seminal contribution of Trivers & Willard (1973), suggests that prenatal shocks may increase fetal loss and reduce the number of boys relative to girls at birth. This has been largely ignored in the economics literature and could affect the interpretation of estimates of the effect of prenatal shocks and that of gender in other applied economics contexts. This paper analyzes the effect of *in utero* exposure to a shock - civil conflict in Nepal - on (i) fetal loss, and (ii) gender and (iii) health at birth. Maternal fixed effects estimates show that exposed pregnancies are more likely to result in a miscarriage and in a female birth, but exposed newborns are neither smaller nor more subject to neonatal mortality.

JEL codes: I10, J13, O15
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1 Introduction

There is now a sizeable economics literature interested in the effect of health shocks experienced *in utero* on later health and socioeconomic outcomes (see Almond & Currie (2011) for an excellent recent survey). In contrast to the issue of selection through early-life mortality, mortality selection *in utero* has so far been largely ignored. Understanding this selection mechanism could shed new light on estimates of the impact of prenatal health shocks. One particularly intriguing aspect of prenatal selection is known as the Trivers-Willard hypothesis (TW hereafter). Trivers & Willard (1973) hypothesized that, in a number of species including humans, females should have evolved to favor offspring of a particular gender according to their circumstances. As predicted by this hypothesis, there is evidence that adverse maternal health shocks around conception or during pregnancy lead to a sex ratio skewed in favor of girls. If exposure to maternal stressors *in utero* influences the sex ratio, this may mediate some of the effects of exposure to these stressors on later life outcomes such as cardio-vascular disease and income, which are correlated with gender.

This paper analyzes the effect of *in utero* exposure to a maternal stressor in the form of the recent civil conflict in Nepal, and makes two novel contributions to the literature. It is the first study to provide maternal fixed-effects estimates of the impact of an environmental stressor during pregnancy on fetal loss and on gender, so that findings are robust to changes in maternal composition correlated with the shock of interest. In addition, it is the first to produce a test of the effect of a maternal stressor on (i) fetal loss, (ii) gender at birth and (iii) *in utero* selection on unobserved health in a unified setting, and therefore to shed light on all dimensions of the TW hypothesis to obtain a more robust and fuller picture.

Accurate data on fetal loss are difficult to obtain as data collection needs to start before many women know they are pregnant, and in retrospective surveys, women are likely to underreport miscarriages even when they were aware of having been pregnant. Twelve to 15 percent of clinical pregnancies in developed countries end in a miscarriage,[1] while self-reported miscarriage rates based on retrospective surveys for a range of developing countries vary between 4.6 and 17.8%.[2] Given these sizeable prevalence rates, prenatal shocks such as war and economic crises may generate substantial selection along unobserved fetal health.

Trivers & Willard (1973) hypothesized that women should favor sons

[1] A pregnancy is defined as clinically recognizable from 6 weeks after the last menstrual period onwards (about 4 weeks from conception). Reported rates of fetal loss including pregnancies of less than 4 weeks range from 17 to as much as 89 percent of all conceptions (Nepomnaschy et al. 2006).

[2] Casterline (1989); Garcia-Enguidanos et al. (2002); Nepomnaschy et al. (2006).

when in good condition and favor daughters when in poor condition in order to maximize the number of surviving grandchildren. This follows from the assumption that the variance of reproductive success (i.e., quantity and quality of offspring) is higher for males than for females. The reproductive success of males could have higher variance than that of females for a number of reasons. The archetypical example is that of a polygynous society where prospects on the marriage market are driven by rank in society (e.g., based on wealth or health status). In this context, low-rank families would be less likely to marry their sons than daughters and therefore they should favor daughters over sons in order to ensure the continuation of the family. On the contrary, high-rank families would prefer to have sons, as these sons would likely have more than one mate. Even in societies that are not polygynous today, biological mechanisms developed during evolutionary time could have persisted.

There is indeed evidence that adverse maternal health shocks around conception or later in pregnancy lead to a sex ratio skewed in favor of girls. Such a phenomenon has been recorded in a range of circumstances, including earthquakes (Fukuda et al. (1998), Torche & Kleinhaus (2012)), terrorist attacks (Catalano et al. 2006), unusually cold years (Catalano et al. 2008), pollution exposure (Sanders & Stoecker 2011), and maternal fasting (Almond & Mazumder 2011). Lack of data on pregnancies not ending in a live birth has prevented previous studies from investigating directly the impact of adverse shocks on fetal loss. In the absence of such data, the "diagnosis" of fetal death from observing a correlation between exposure after conception and gender at birth can only be residual, and when exposure around conception is found to affect the sex ratio, it is not possible to know whether this is due to changes in the sex ratio at the time of conception or due to gender-biased fetal loss.

Importantly, alternative mechanisms could account for the finding that prenatal health shocks alter the sex ratio, with opposite implications on the direction of survivor bias (i.e., selection on unobserved health). Consider an initial distribution of fetal health. Fetuses survive until birth provided that their health endowment exceeds a given survival threshold. A worsening of maternal condition during pregnancy can deteriorate fetal health, thus shifting the fetal health distribution to the left ("scarring" mechanism), or shift the survival threshold to the right (positive selection or "culling" mechanism), or both. Given that males are over-represented in the lower part of the fetal health distribution (see Kraemer (2000), Catalano & Bruckner (2006) and references therein), both mechanisms would lead to a decrease in the sex ratio defined as the number of males relative to females. Both the culling and scarring mechanisms are plausible and have

been shown to be empirically relevant (see Catalano et al. (2008) for culling and Almond & Currie (2011) for scarring). However, the scarring mechanism implies a worsening of health conditional on birth, while the culling (TW) mechanism implies an improvement of health conditional on birth (Catalano & Bruckner 2006). This both motivates further testing of the TW mechanism, since implications for the analysis of prenatal shocks and for the treatment of gender in econometric models differ between the two mechanisms, and provides a test for identifying the mechanisms at work.

I find that civil conflict experienced during pregnancy increases both the probability of spontaneous abortion and the probability that a newborn is female, holding maternal unobserved heterogeneity constant. Conditional on live birth, survivors are not systematically in better or worse health as measured by size at birth and neonatal mortality. This suggests that the skewing of the sex-ratio follows from both scarring and selection mechanisms.

The rest of the paper is organized as follows. Section 2 gives an overview of the TW hypothesis literature. Section 3 depicts the conflict background and discusses aspects of the conflict relevant to pregnancy outcomes. Section 4 presents the data and identification strategy. Section 5 reports the results on pregnancy resolution and gender. Section 6 tests the (TW) selection hypothesis and discusses implications for applied econometric models. Section 7 checks the robustness of these findings, and Section 8 concludes.

2 Trivers-Willard: Assumptions, Evidence, and Interpretation

2.1 Necessary Conditions for the Trivers-Willard Hypothesis

The assumptions underlying the argument in Trivers & Willard (1973) are that: (i) the condition of the offspring at the end of the period of parental investment is correlated with that of the mother during the period of parental investment, (ii) differences in the condition of the offspring at the end of the period of parental investment persist into adulthood, and (iii) male reproductive success is more variable than that of females.

There is abundant evidence that famine, disease, and adverse economic conditions affecting pregnant women and infants have negative consequences on an individual's health and socio-economic outcomes in adult life (Almond (2006); Van den Berg et al. (2006); Maccini & Yang (2009); Almond & Currie (2011)). In addition, there are a number of reasons why male reproductive success could depend more on rank (be it in terms of health or

socioeconomic status) for men than women. As explained in the introduction, in polygynous societies in which marriage prospects depend on rank in society, high-rank males will reproduce more than females and low-rank males even if males and females seek the same traits in mates - e.g., health, wealth. Lazarus (2002) lists several other mechanisms through which the reproductive success of males may be more variable with parental condition than that of females. For instance, in monogamous societies, male reproductive success may also be more dependent on high rank if women seek high-status traits in mates more than men do, as has been observed to be the case, e.g., in 1960s United States (Trivers & Willard 1973). However, Trivers & Willard (1973) also acknowledge that the application of their model to humans is complicated by other influences. Paternal investments in their young is much higher among humans than among most other species. This implies that more paternal time and resources are expended for each offspring than in other species, thus limiting the number of children that even the most sought-after male can have and hence reducing the variability of male reproductive success. Over and above the question of the applicability of the TW hypothesis to human societies due to their complex social structure, there is controversy over the likelihood of parental manipulation of offspring gender to have evolved in high vertebrates, including humans (Krackow 2002).

2.2 Evidence of Decreased Secondary Sex-ratio Following *in utero* Exposure to Maternal Stressors

Notwithstanding controversy over the interpretation of these findings, a number of studies provide tests of the effect on the sex-ratio at birth of comparatively "time-invariant" maternal condition, as well as of environmental stressors experienced around conception and during pregnancy.

In US natality data covering 48 million births, Almond & Edlund (2007) find that the sex-ratio at birth is lower- and that the proportion male among infant deaths is higher- among unmarried mothers, which is consistent with the TW hypothesis. This echoes findings from a smaller, survey-based sample in which a lower sex-ratio was found among mothers who did not live with a male partner before conception (Norberg 2004). However, the findings of the literature taken as a whole are mixed (Lazarus (2002); Grant (2009); James (2012)).[3]

[3]Lazarus (2002) mentions 54 studies considering the effect of parental socioeconomic status on sex-ratio, and reports that 26 of these studies support the hypothesis. However, Lazarus (2002) emphasizes the difficulty in concluding due to the fact that a number of studies have small sample sizes (some of only a few hundreds). Indeed, in a meta-analysis

There is more consistent evidence of a sex-ratio skewed in favor of girls resulting from adverse maternal health shocks around conception or during pregnancy. Such a phenomenon has indeed been recorded in a range of circumstances, including earthquakes (Fukuda et al. (1998), Torche & Kleinhaus (2012)), terrorist attacks (Catalano et al. 2006), unusually cold years (Catalano et al. 2008), pollution exposure (Sanders & Stoecker 2011), and maternal fasting (Almond & Mazumder 2011). But to the best of my knowledge, no previous study has estimated the effect of a prenatal shock on both the sex ratio and fetal loss. Some information on fetal loss can be obtained indirectly through the timing of exposure. If exposure during the first month is found to matter (as in Fukuda et al. (1998) and Almond & Mazumder (2011)), then it is not clear whether the effect on the sex ratio at birth is due to an effect on the sex ratio at conception or due to gender-differentiated rates of miscarriage.[4] When an effect of exposure later in the pregnancy is found, as in the case of Torche & Kleinhaus (2012) (third month) and Catalano et al. (2006) (fifth month), then it suggests that the sex ratio is altered via fetal loss. But in the absence of data on pregnancies that do not end in a live birth, the "diagnosis" of fetal death can only be residual.

The interpretation of this evidence as the manifestation of a TW mechanism is debated. In the next section, I present a conceptual framework allowing me to derive testable implications of the TW hypothesis beyond the predicted effects on the secondary sex-ratio (i.e., the sex ratio at birth).

2.3 Survival Advantage Interpretation

Despite the substantial body of evidence showing that maternal stressors experienced between conception and mid-pregnancy tend to decrease the sex-ratio at birth, the interpretation of such sex-ratio skewing as evidence of an evolved ability of mothers in poor condition to adjust (unconsciously) their offspring's sex-ratio *à la* TW remains highly controversial. According to Catalano & Bruckner (2006), a test of the dominance of selection versus scarring effects provides a test of the TW interpretation. The tension between scarring and selection is well-known in the demographic and economic literature concerned with the effect of health shocks experienced *in utero* (Almond 2006) or in the early years of life (Bozzoli et al. 2009) on adult outcomes, although it has tended to focus on selection on mortality

of tests of the effect of rank on sex of offspring among non-human primates, Brown & Silk (2002) conclude that the magnitude of the effects decrease as sample sizes increase and therefore that "the observed effects could be the product of stochastic variation in small samples." (p. 11252).

[4]See Lazarus (2002) and Fukuda et al. (1998) for discussions on the determination of the sex-ratio at conception.

after birth. Here I summarize the arguments and provide a succinct algebraical and graphical illustration to clarify the discussion of the expected effect of exposure to conflict *in utero* in the context of gender-differentiated fetal mortality.

Let h_i^* be the unobserved health endowment of individual i *at conception*, which, in an ideal health environment, is distributed according to the density $f(h_i^*)$ and a cumulative distribution $F(h_i^*)$. Assuming further that there is a survival threshold d_0 below which fetuses cannot survive to birth, the mean health endowment at birth is:

$$E(h_i^*|h_i^* > d_0) = \frac{\int_{-\infty}^{\infty} h_i^* g(h_i^*)\,\mathrm{d}h_i^*}{1 - F(d_0)} \tag{1}$$

where $g(.)$ denotes the (truncated) density function of health at birth, $g(h_i^*) = f(h_i^*)\ \forall h_i^* > d_0$ and $g(h_i^*) = 0$ otherwise. On the one hand, a health shock experienced *in utero* may deteriorate fetal health, leading to a shift to the left of the distribution of the health endowment *at birth via* a scarring mechanism. As pointed out by Almond (2006), an increase in mortality may arise from this shift (from m_0 to m_0^- in Figure 1), but such an increase in mortality could not result in an improvement of the health distribution at birth, since for all $h > d_0$, the probability of observing a birth with $h_i^* < h$ increases.

On the other hand, the survival threshold d_0 may shift right to d_1 if the negative health shock *in utero* is such that a better health endowment at conception is now required to survive to birth, and thus positive selection (or "culling") occurs - i.e., the average health endowment at birth is higher following the shift of the survival threshold. In this case, mortality *in utero* goes up to m_1 (m_1^-) in the absence (presence) of scarring.

Assuming that both scarring and selection mechanisms occur, the mean health endowment at birth after the negative *in utero* health shock would become:

$$E(h_i^*|h_i^* > d_1) = \frac{\int_{-\infty}^{\infty} h_i^* g^-(h_i^*)\,\mathrm{d}h_i^*}{1 - F^-(d_1)} \tag{2}$$

where $f^-(h_i^*)$ ($F^-(h_i^*)$) is the density (cumulative) distribution function following the leftward shift of the initial distribution of the health endowment, $g^-(h_i^*) = f^-(h_i^*)\ \forall h_i^* > d_1$ and $g^-(h_i^*) = 0$ otherwise.

The total effect of the scarring and selection effects on the distribution of the fetal health endowment *at birth* depends on the magnitude of the leftward shift in the health endowment distribution relative to that of the right shift of the survival threshold, which is an empirical question.

What are the implications of the above discussion in terms of the TW hypothesis? There is evidence that males outnumber females among fe-

tuses who die *in utero* (Byrne et al. (1987); Rueness et al. (2012)), and that sex-specific fetal death rates are higher among males (Møller (1996); Mizuno (2000)), suggesting that there are more males in the lower portion of the health distribution. Therefore, a larger number of male fetuses would be spontaneously aborted under both the "scarring" and the "selection" cases, leading to a decrease in the sex-ratio in both cases. However, only the skewing of the sex-ratio stemming from the selection mechanism can be attributed to the TW hypothesis (Catalano & Bruckner 2006).[5] Empirically, we should observe a decrease in the sex-ratio at birth under either (or both) mechanism(s), but health at birth should worsen if scarring dominates, and improve if selection dominates, or be unchanged if both mechanisms cancel each other out. Another testable implication of the "selection" hypothesis is that the absolute improvement in the mean endowment of surviving male fetuses should be larger than that for females because assuming that the distribution of male fetal health lies to the left of that of females implies that the mean male fetal health endowment between the old and new survival thresholds is lower.

3 Civil Conflict in Nepal as a Maternal Stressor

The prenatal shock of interest in this paper is exposure to civil conflict in Nepal. More than half of all countries have experienced at least one episode of civil conflict since 1950 (Blattman & Miguel 2010), making it an unfortunately common source of damage to mothers' physical and emotional health, especially in developing countries. In this section, I describe the nature of the Nepalese conflict and summarize the implications of civil conflict for maternal health. I then review the literature on the impact of social conflict on fetal loss, sex ratios and health at birth and situate the present paper in this literature.

3.1 Civil Conflict in Nepal

Nepal became a parliamentary monarchy in 1990. Despite the multiparty democratic elections that followed, a Maoist insurgency broke out in 1996, only to end in 2006. The insurgency started in February 1996 in the Rolpa

[5]Some theorists have argued that the higher frailty of males has itself been favored by natural selection because it increases the probability that mothers will have sons when in good condition and girls when in bad condition (Wells 2000). Under this assumption, both the scarring and selection mechanisms could be interpreted as consistent with TW. However, the motivation for distinguishing between the relative importance of the two mechanisms (i.e., that they have different implications for research on the effect of prenatal shocks and on the effect of gender) remains intact.

district. At first, it was concentrated in a few Communist strongholds in Western Nepal, but by the end of the war, conflict-related casualties were recorded in 73 out of the 75 Nepalese districts. The Maoists' presence varied across districts from sporadic attacks to the organization of their own local governments and law courts, resulting in wide geographic variation in conflict deaths (Figure 2).[6] Over the course of the conflict, Maoists attacked government targets such as army barracks, police posts, and local government buildings (Do & Iyer 2010). They were also reported to terrorize, loot, abduct, and physically assault civilians (Human Rights Watch (2004); Bohara et al. (2006)). On the other hand, government security forces also killed civilians and were accused of torturing, displacing and summarily convicting civilians (Bohara et al. 2006).

A crucial moment in the conflict was the ending by the Maoists of a short-lived cease-fire in November 2001. From then on, the government's response intensified dramatically, involving the Royal Nepal Army, leading to an escalation of violence (see Figure 3). Building on opposition to King Gyanendra's authoritative reaction to the prolonged conflict, the Maoists joined forces with some of the country's major political parties, leading to the signing of a peace agreement in November 2006 and the creation of an interim government led by a power-sharing coalition including the Maoists. This put an end to a decade of conflict that led to the deaths of over 13,000 people (Informal Sector Service Center 2009).

3.2 Civil Conflict and Maternal Condition

There are a number of potential mechanisms through which maternal physical or psychological health could be negatively affected by civil conflict, among which: malnutrition due to the destruction of sources of income and crops; psychological stress; poorer access to health care due to travel restrictions, destruction of infrastructure, and lower income; increased disease prevalence; and the direct effect of physical violence. It is difficult to pinpoint the relative importance of each of these sources of maternal stress, broadly defined as a worsening of maternal condition, in the effect of conflict in the case of Nepal. However, given the common occurrence of civil conflict, the effect of such an event is arguably of interest in itself.

I hypothesize that civil conflict experienced *during* pregnancy should

[6]Several arguments have been put forward to explain the district variation in the intensity of the insurgency, including geography (Murshed & Gates (2005); Bohara et al. (2006); Do & Iyer (2010)), poverty (Murshed & Gates (2005); Do & Iyer (2010)), lack of political participation (Bohara et al. 2006), and inter-group inequality (Murshed & Gates (2005); Macours (2011)). Given that these variables are likely to also affect fetal and neonatal health, it is important to control for district heterogeneity in the analysis and check the robustness of findings to potential confounders, as discussed in Sections 4.2 and 7.

decrease the sex-ratio, at least in part as a result of higher fetal loss, and that health at birth may be affected either positively or negatively, depending on the relative strength of the culling and scarring mechanisms.

Studies testing for changes in the sex-ratio following recent wars have found no effect (Polasek et al. 2005) or found a decrease in the sex-ratio (Zorn et al. (2002); Ansari-Lari & Saadat (2002)) when comparing the (unconditional) odds ratio of being male during the war compared to before- and after the war.[7]

Contrary to the above studies, I also estimate the effect of exposure to conflict *in utero* on fetal loss, which sheds light on the contribution of fetal loss (as opposed to changes in the primary sex ratio) to changes in the secondary sex ratio, and increases confidence in the estimates on the effect of civil conflict on the sex ratio.

Few studies have considered the effect of stressful economic shocks, war, and social crisis on the incidence of fetal loss. Rajab et al. (2000) compare the incidence of spontaneous abortions in the 5 years before and the 5 years after the Gulf War based on hospital records from the main referral hospital of Bahrain, and find an increase in referrals in the post-war period. Using Chinese retrospective fertility histories data, Cai & Feng (2005) find that the probability of miscarriage and stillbirth increased in China during years corresponding to the Chinese Cultural Revolution and years corresponding to the famine caused by the Great Leap Forward, after controlling for a linear time trend and parental socioeconomic characteristics. In this paper, I provide a tighter test of the effect of violent events on fetal loss by exploiting geographical variation in exposure to conflict, using monthly conflict event data, and controlling comprehensively for unobserved maternal heterogeneity using mother fixed-effects.

Using similar techniques to those used here, two papers have estimated the impact of conflict-related events on birth weight. Camacho (2008) found that, in Colombia, landmine explosions taking place in the mother's municipality during the first trimester of pregnancy had a small negative impact on birth weight. Mansour & Rees (2012) similarly found that first-trimester conflict casualties increased the probability of low birth weight

[7]Somewhat surprisingly, a literature documenting an *increase* in the sex-ratio following the two World Wars has developed in parallel to that on the TW hypothesis (see Grant (2009) for a review). Grant (2009) proposes an explanation reconciling the findings for the two World Wars with the literature finding the reverse effect of prenatal stressors, including wars, on the secondary sex ratio. She suggests that maternal stress could raise the production of testosterone, which both increases the likelihood of conception of a male if experienced *before* conception and increases the probability of spontaneous abortion of males if experienced *after* conception. In this paper I focus on exposure during the nine months preceding birth and so I expect to observe a *decrease* in the sex ratio.

during the second Intifada in the Palestinian West Bank.[8] Both findings are robust to the inclusion of maternal fixed effects, and both sets of authors argue that maternal stress is the most likely explanation for their findings. However, neither study considers gender or fetal loss outcomes.

4 Data and Identification Strategy

In the first part of this section, I describe the data used in the analysis. I first present the fertility histories dataset, including a detailed description of how key variables were constructed and how the sample used in the analysis was obtained. I then describe the source of data on conflict casualties, and provide descriptive statistics for the variables used in the analysis. In the second part of the section, I present the econometric models used in the analysis, and explain how these address concerns regarding the causal identification of the effect of prenatal exposure to civil conflict, namely: parental selection due to pregnancy decisions, parental selection on biological characteristics, and unobserved heterogeneity in reporting pregnancy outcomes and female births.

4.1 Data

Demographic and Health Surveys (DHS) have been carried out in a number of developing countries as part of the Measure DHS project, a worldwide USAID-funded project aimed essentially at providing detailed, reliable information on fertility, family planning, maternal and child health and mortality.

The second and third DHS carried out in Nepal took place in 2001 and 2006, respectively.[9] The DHS surveys took place either before or after the most intense conflict period, so that data collection was not greatly disrupted, although in 2001, six out of 257 sampling units had to be dropped from the sample for security reasons (MOHP, New Era and ORC Macro (2002), p.6). Both surveys collected data from a nationally representative sample of women aged between 15 and 49 (if ever married in the case of the 2001 survey). Respondents were asked about their entire fertility history, including dates of all births and deaths of any liveborn child and dates of

[8] Mansour & Rees (2012) also find that exposure to conflict later in pregnancy may increase the probability of low birth weight, but these findings are less consistent across specifications.

[9] Data from the 1996 and 2011 DHS were not included in the analysis because the preferred specification (mother fixed-effects) requires variation in conflict exposure within mother. There is no such variation for women interviewed in 1996 (when the conflict started) and for children born within the 2011 DHS "calendar period" (for whom size at birth information was collected), as the conflict ended in 2006.

end and duration of all other pregnancies.[10] The questionnaires contain a number of probes for these, and enumerators were specifically trained to ensure that this information, that is central to the survey, is reliable.[11] These fertility histories are used here to create a panel dataset where mothers are the cross-sectional units and pregnancies the "longitudinal" unit, as in Bhalotra & van Soest (2008).

Due to the retrospective nature of the data, there may be measurement error in the dependent variable. Using Malaysian data from the 1970s and 1980s, Beckett et al. (2001) find that recall error in fertility histories is not an issue for live born children, except for some age heaping (e.g., rounding at one year old for children who die when 11 or 13 months old). As a consequence, I allow for age heaping such that the neonatal mortality indicator switches on for children who were reported to be up to one month old at the age of death.[12] More importantly, I also address this issue by restricting the analysis to children born less than 5 years before the start of the survey.[13] This coincides with the "calendar" period of the survey, and Becker & Sosa (1992) show that the use of a calendar improves the quality of data collected in retrospective demographic surveys.

Data on pregnancies that do not result in a live birth are prone to more measurement error, especially in the form of underreporting (Beckett et al. 2001). By restricting the sample to the five years preceding each survey, underreporting should be substantially reduced.

Different women were interviewed in the 2001 and 2006 DHS surveys. However, the degree of measurement error in the reporting of miscarriages can be appraised by comparing the average rate of miscarriage obtained for children conceived in a given (Nepali) calendar year, but reported by different mothers 5 years apart (in 2001 and in 2006), as depicted by Figure 4. The figure shows that, for the recent period covered by the data used in this paper, average miscarriage rates are reasonably consistent across surveys, especially considering that the rates are based on comparatively small year samples and for different women. It is also worth noting the sharp increase in miscarriages coinciding with the conflict escalation.

By pooling the 2001 and 2006 DHS cross-sections of mothers, I obtain a sample of children aged below five at the time of either survey whose dates

[10] In these surveys, women were asked to report each of their pregnancies in turn, and, one by one, whether the baby was "born alive, born dead, or lost before birth". If they answered either of the two last options, the respondents were then asked about the month and year the pregnancy ended and its duration. If the child was born alive, I assume the duration of the pregnancy to be 9 months as I only observe the date of birth.

[11] See MOHP, New Era and Macro International Inc. (2007) for more information.

[12] Strictly speaking, neonatal mortality relates to mortality in the first 4 weeks of life.

[13] In the case of pregnancies that do not end in a live birth, the sample is restricted to pregnancies starting less than 5 years and 9 months before the start of the survey.

of birth span the whole period of the conflict, namely 1996-2006.

There are 14,107 pregnancies that started no more than 68 months before the month of interview. I restrict the analysis to singletons, as is standard in the demographic literature (dropping 201 pregnancies). I drop 132 pregnancies starting less than 9 months before the date of interview since, for this time period, only pregnancies that do not end in a live birth are recorded in the data. I also drop 771 pregnancies of women who are visiting the household. I then restrict the analysis to children who were conceived in the place where their mothers were interviewed, in order to limit measurement error in exposure to conflict. This drops 1116 pregnancies, but my findings are not sensitive to including these children (see Section 7). The resulting sample used for the analysis of pregnancy resolution (miscarriage and stillbirth) contains 11,887 *pregnancies*. In order to focus on biological mechanisms, I drop 315 pregnancies for whom women report "having done something to end the pregnancy" or refuse to answer the question on whether or not they have done something to end the pregnancy, yielding a sample of 11,572 pregnancies including 596 miscarriages, 130 stillbirths and 10,846 live births. For the analysis of outcomes observed only at *birth*, I further drop the pregnancies that did not end in a live birth (726), children who were not born at least a full month before the interview and therefore were not fully exposed to neonatal mortality risk (244), and those whose mothers did not report their (subjective) size at birth (11). The resulting *live births* sample counts 10,591 children.[14]

A number of outcomes are considered, namely binary indicators for miscarriage, stillbirth, and, conditional on live birth, gender, size of baby at birth as reported by the mother, and neonatal mortality.

Following the existing literature, miscarriages and stillbirths are studied separately. In developing countries, this distinction may be particularly relevant since stillbirths occurring during delivery contribute a large share of stillbirths, and risk factors in these intrapartum deaths are different from those involved in pre-labor fetal deaths (McClure et al. 2006*a*).

For each pregnancy, mothers are asked whether the baby was "born alive, born dead, or lost before birth". If they answer that the baby was born dead or lost before birth, mothers are then asked whether they or someone else had done "something to end this pregnancy" (MOHP, New Era and Macro International Inc. 2007). The miscarriage (stillbirth) variable is equal to one if the mother answers that the baby was "lost before birth" ("born dead") without any action taken to end the pregnancy, and

[14] As a point of comparison, Mansour & Rees (2012) use a sample of 1,224 births, Zorn et al. (2002) a sample of 4,966 births affected by war, Rajab et al. (2000) a sample of 14,850 pregnancies, Camacho (2008) a sample of 781,000 births, Polasek et al. (2005) a sample of 861,516 births, and Cai & Feng (2005) a sample of 1.3 Million pregnancies.

zero if the child was born alive. When the mother answered that some action was taken to end the pregnancy, or when she gave no answer about intent, the miscarriage and stillbirth indicators are set to missing in order to focus on biological mechanisms.[15]

Although newborn health is difficult to capture with a single variable, birth weight is a commonly used measure. In a country like Nepal, where over 80 percent of babies are born at home (MOHP, New Era and Macro International Inc. 2007), birth weight is unknown for the majority of children. However, the DHS asked mothers to report whether at birth the child was 'very large', 'larger than average', 'average', 'smaller than average' or 'very small' at birth. I use this information to create a 'small baby' dummy equal to one if the child was 'smaller than average' or 'very small', and zero otherwise.

I then merge the individual data from the pooled Nepalese DHS with the number of conflict deaths per month per district of Nepal compiled by the Informal Sector Service Center (INSEC). INSEC is an independent, well-regarded, human rights NGO based in Kathmandu and with representatives in each of the 75 Nepalese districts, who monitor human rights violations. Data from INSEC has been extensively used in the media, international agencies and government reports, and in a number of academic studies, including Bohara et al. (2006) and Do & Iyer (2010). All conflict exposure variables are expressed in deaths per 1000 district inhabitants as per the last pre-conflict census (1991).

Summary statistics for all the variables used in the analysis are reported in Table 1. For the purpose of this table (but not in the analysis), the sample is divided into three groups of districts, based on the district's position in the distribution of total district deaths (per 1000 inhabitants) over the conflict period, as in Figure 2. Fertility is lower in the low conflict-intensity tercile, as illustrated by the lower proportion of children of pregnancy order five and above in this district group. Children born in the low conflict-intensity tercile are more often born in urban areas, and to educated mothers. However, children in the low conflict-intensity tercile are less often born to parents from the more privileged classes (Brahmin and Chhetri), more likely born to somewhat less well-off other Tarai/Madhesi castes and Muslim parents, and less likely born to parents from indigenous groups (Janajati). They are less likely to be small babies at birth. However, there is no apparent difference in the probability of neonatal death across

[15]Similar results were obtained when miscarriage and stillbirths were instead defined according to whether the pregnancy resulted in an unintended fetal death occurring by the sixth month of pregnancy (miscarriage) or afterwards (still birth). In the context of developing countries, a late gestational threshold for defining fetal loss as stillbirth is particularly justified because survival is unlikely for children born at gestational ages below 28 weeks (approximately 1000g) (McClure et al. 2006*a*).

conflict-intensity groups, and no clear cross-sectional correlation between overall conflict intensity and the likelihood of a female birth, a miscarriage or a stillbirth. Unless otherwise specified, the magnitude of the estimated effects are interpreted in relation to the change in the outcome of interest when going from the mean conflict exposure in the low conflict-intensity tercile to the high conflict-intensity tercile (e.g., in the case of exposure *in utero*, for a change of $0.011 - 0.002 = 0.009$ average monthly casualties per 1000 inhabitants).

As mentioned in Section 3.1, the conflict started in 1996 and remained localized to a minority of districts for some years (Figure 5, top-left panel), remaining of low intensity where fighting was taking place (Figure 5, top-right panel), and slowly spreading to other districts over time until the escalation of 2001, at which time all but the two small districts of Manang and Mustang experienced at least some violence. The peak in conflict intensity was experienced in 2001-2003, after which the number of casualties started to decrease (Figure 3 and Figure 5, top-right panel). Therefore, the 1996-2000 period is, in most districts, a pre-conflict period, and the end of the period covered by the data corresponds to a period of lower conflict intensity. The graph in the bottom left (right) panel of Figure 5 shows, year by year, the number of districts where pregnancies were, on average, exposed to less (more) conflict in that year compared to the previous year. These two last graphs illustrate the fact that, throughout the 1999-2006 period, conflict intensity was at times increasing in some areas and decreasing in others, thus providing arguably exogenous variation in exposure to conflict intensity *in utero* at various points in space and time. For the overall sample of pregnancies used in the paper, the mean number of deaths per month during the gestation period is 0.0055 (minimum: 0 and maximum:.484), with a standard deviation of 0.0138 and a median of 0.00054 - which is much lower than the mean, as expected from the above description of the pattern of conflict.[16]

4.2 Identification Strategy

I test three hypotheses based on existing evidence and the conceptual framework of Section 2.3. Namely, I test whether *in utero* conflict exposure (i) increases the probability that a child born alive is female (i.e., decreases the sex ratio), (ii) increases the probability of fetal loss, and (iii) results in scarring or positive selection on fetal health, or both. I expect to observe an increase in the probability of a child being female, and an increase in fetal loss. The third test is then needed in order to decide if a culling (TW)

[16]The median number of casualties per district was 145 across the 75 districts over the whole conflict period.

interpretation is warranted. This does not necessarily follow from (i) and (ii) since the scarring effect of maternal exposure to conflict could account for both an increase in fetal loss and a decrease in the sex ratio, independently of any evolved adaptation of the fetal survival threshold to maternal condition.

I start by estimating linear panel data fixed-effects models of the form:

$$\begin{aligned} Y_{imdt} =& \beta_0 + \beta_{pre} pre_{dt} + \beta_{utero} inUtero_{dt} \\ &+ X'_{imdt}\beta_X + Z'_{mdt}\beta_Z + Y'_t\beta_y + D_d + u_{imdt} \end{aligned} \tag{3}$$

where Y_{imdt} is one of several outcomes of interest for child i of mother m conceived in district d and in month t; pre_{dt} is the *cumulated* number of (district) conflict deaths that have occurred up to the month before the conception of the index child conceived in district d in month t; $inUtero_{dt}$ is the *average monthly* number of casualties during pregnancy (so as to be comparable between pregnancies with different gestational periods); X_{imdt} are pregnancy-specific demographic controls, namely age of mother at conception and its square, pregnancy order indicators (for second, third, fourth, and "five and above"), and 11 calendar month of conception dummies. Z_{mdt} are pregnancy-invariant maternal characteristics, namely dummy variables for urban residence, maternal education, and caste. Y_t is a set of year of conception dummies, D_d a set of district fixed effects, and u_{imdt} is an error term assumed to be independent between districts but not necessarily within district, and robust to heteroskedasticity of an arbitrary form.

The coefficient of interest is β_{utero}. The coefficient β_{pre} is not given a causal interpretation, but in the main regressions, pre_{dt} is included as a control for potential time-varying unobserved heterogeneity correlated with both changes in district conflict intensity over time and changes in the dependent variable.

I then exploit the fact that there is substantial variation in prenatal exposure to conflict within mother and estimate the following mother fixed-effects specification:

$$\begin{aligned} Y_{imdt} =& \gamma_0 + \gamma_{pre} pre_{dt} + \gamma_{utero} inUtero_{dt} \\ &+ X'_{imdt}\gamma_X + Y'_t\gamma_y + M_m + v_{imdt} \end{aligned} \tag{4}$$

Estimates obtained with this equation are robust to selection on unobserved characteristics of women who become pregnant or give birth at times of higher conflict intensity. For clarity, in each mother fixed effects specification, I explicitly restrict the relevant samples to children with at least another sibling in the sample, and refer to this sample as the "siblings" sam-

ple. In an intermediate step between the district fixed-effects analysis and the maternal fixed-effects analysis, I estimate district fixed-effects models on the siblings sample in order to establish whether the effect of prenatal conflict exposure systematically differs for families who have at least two children in the five years preceding the survey compared to the rest of the sample when I do not control for mother unobserved heterogeneity.

Pregnancy-specific characteristics X_{imdt} are included in all the main regressions. Pregnancy order and maternal age are indeed well-known determinants of obstetric outcomes, they are correlated with child gender (Lazarus 2002), and pregnancy order has been found to have a large effect on parental investments in child quality (Black et al. 2005), and thus could influence prenatal health investments. The inclusion of these control variables should therefore increase the precision of my estimates. In addition, given the structure of the data (pregnancy histories), maternal age and pregnancy order increase over time, and this might bias the estimates of the impact of conflict if not controlled for. Similarly, there may be seasonal effects relevant both for conflict intensity and health outcomes, justifying the inclusion of month of conception dummies. As discussed in Section 7, results are robust to excluding these controls.

The preferred specification is the mother fixed-effects specification, for three reasons: parental selection due to pregnancy decisions, parental selection on biological characteristics, and unobserved heterogeneity in reporting pregnancy outcomes and female births.

Although the month-to-month variation in conflict casualties would have been difficult for parents to predict, they may have had some information at their disposal which would have allowed them to partially predict future conflict intensity. If this were the case, then conflict intensity during pregnancy could have influenced pregnancy decisions in a way that could be correlated with unobserved parental heterogeneity that matters for the outcomes of interest in this paper. For instance, if health-conscious parents were more likely to postpone a pregnancy in anticipation of increased conflict intensity in the near future, and if health-conscious mothers were also less likely to experience a stillbirth due to better delivery conditions, then conflict intensity could be positively correlated with the probability of a stillbirth due to a change in the composition of mothers. I test for changes in the composition of mothers by estimating Equation 3 using as dependent variables a range of observable maternal characteristics and excluding Z_{mdt} from the set of regressors. Results in Table 2 suggest moderate selection on *observable* characteristics. Some high-caste mothers (Brahmin or Chhetri) and the very few mothers with university education are comparatively less likely to become pregnant after they have been exposed to more conflict

or when they anticipate more intense conflict during their pregnancy. This finding is in line with that uncovered by Agadjanian & Prata (2002) during the civil conflict in Angola, where better educated and higher SES women appear more likely to have decreased fertility during the conflict.[17]

A different type of parental selection may arise due to biological mechanisms rather than intent. Nepomnaschy et al. (2004) find evidence that everyday psychological stress reduces female reproductive ability, while Subbaraman et al. (2010) propose that more reactive women, defined as exhibiting "autonomic, immune and neuroendocrine responses at relatively low levels of provocation, and [having] a stronger response than others to stimuli that provoke responses in most individuals" (pp.2085), are more likely to spontaneously abort less fit fetuses among whom males are over-represented. It would seem likely that more reactive women are also less likely to conceive under stress, so that not controlling for unobserved reproductive heterogeneity could lead to an underestimation of the effect of conflict intensity on both the probability of fetal loss and on the probability of a female birth.

Finally, given the self-reported nature of the pregnancy histories, reporting bias could be correlated with exposure to conflict in a direction that is unclear *a priori*. Reporting bias is likely to be non-negligible for pregnancies that do not end in a live birth and for subjective size at birth. In addition, in a country such as Nepal, where a number of parents prefer sons to daughters (Arnold 1997), there may be a degree of sex-specific reporting bias such that some mothers are more likely to report the birth of a girl than others.

5 Effect of Conflict on Gender and Pregnancy Resolution

Table 3 reports estimates of the effect of exposure to conflict *in utero* on female gender. This and the following tables are arranged as follows. In the first column, I estimate Equation 3 on the whole sample. In Column

[17] Better-educated and higher-status women may be better informed, or be more likely to make sophisticated family planning decisions. Given the Maoist ideological nature of the insurgency, another plausible explanation for this pattern of selection would be that groups who felt most threatened by the Maoist insurgency postponed (or stopped) having children in reaction to the intensity of violence in their area. The heterogeneous response to conflict intensity in the probability of being pregnant might also reflect the higher vulnerability of lower socio-economic women to rape (and thus unprotected sex). Given the available data, it is not possible to establish the reason for the selection on socioeconomic characteristics observed here. However, the preferred maternal fixed effects specification is robust to any source of selection into becoming pregnant, as long as it is constant across pregnancies for a given woman. I address the potential remaining biases in Section 7.

(2), I restrict the sample to pregnancies with at least another sibling in the relevant time period in order to assess whether families with at least two children conceived within 5 years and nine months of the survey are differently affected by violent conflict compared to the rest of the sample. In Column (3) I present maternal fixed effects estimates (Equation 4), which are robust to selection on time-invariant parental heterogeneity. Column (4) repeats the regression in Column (3) but allowing for different effects of *in utero* exposure according to whether it takes place in the first (starting in the month of conception (mc) and lasting until $mc + 4$) or second half (spanning $mc + 5$ to $mc + 9$) of the pregnancy. Finally, Column (5) allows for different effects of *in utero* exposure by trimester.[18]

When selection on unobservable parental characteristics is not controlled for (Columns (1) and (2)), exposure to conflict does not appear to have a significant effect on sex at birth. However, once I allow exposure to conflict to be correlated with unobservable maternal heterogeneity (Column (3)), results indicate that maternal exposure to conflict during pregnancy significantly increases the probability of a female birth. The direction of maternal selection is consistent with a number of plausible explanations. It could for instance be due to women who are more reactive being less likely to become pregnant at times of more intense conflict (see Section 4.2). Or it could be the case that women who feel that they do not yet have "enough" sons are both more likely to become pregnant irrespective of high conflict intensity and less likely to report the birth of a girl. Going from mean *in utero* exposure in the low-intensity district group to the high-intensity group (i.e., an increase of 0.009 monthly average casualties per 1000 inhabitants) leads to an increase in the probability of a female birth by 1.8 ppt, which is comparable to estimates in Zorn et al. (2002) (1.4 ppt) and in Ansari-Lari & Saadat (2002) (between 1 and 1.7 ppt).

A direct test of the impact of conflict on pregnancy resolution is presented in Table 4, where I estimate the effect of exposure to violent conflict on the likelihood of miscarriage against the alternative of a live birth.

When maternal heterogeneity is controlled for, the probability of miscarriage increases with exposure to conflict *in utero* (Column (3)). Within-district point estimates are also positive, but smaller and statistically in-

[18]The DHS include the month and year of birth (or end of pregnancy in the case of pregnancies that do not end in a live birth), but not the day. For instance, for a child conceived on 1st January, the first trimester would coincide with January, February, and March, while for a child conceived on 31st January, the first trimester would essentially correspond to February, March, April. In order to reflect the uncertainty over when conception occurred between the start and end of the month, first (second) [third] trimester exposure is defined as average monthly casualties during mc to $mc+3$ ($mc+3$ to $mc+6$) [$mc + 6$ to $mc + 9$].

significant (Columns (1) and (2)). The direction of maternal selection is consistent with the hypothesis that women who are more reactive (and thus may be more likely to miscarry (Subbaraman et al. 2010)) are also less likely to become pregnant at times of more intense conflict (see Section 4.2). Or it could be the case that women who have lost a pregnancy are both more likely to try to become pregnant again irrespective of high conflict intensity and less likely to report another fetal loss. Going from mean *in utero* exposure in the low-intensity district group to the high-intensity group leads to an increase in the probability of miscarriage by 0.77 ppt (11.6 percent of the mean).

In addition, as would be expected, in the current sample, 90 percent of miscarriages occur in the first five months of pregnancy. The robustness of the estimated effect of exposure to conflict *in utero* on miscarriage can thus be tested by dividing the pregnancy period along the five month divide. The results are presented in Column (4) and confirm that the effect of conflict exposure *in utero* on both miscarriage (Table 4) and gender at birth (Table 3) is driven by conflict during the first 4 to 5 months following conception, which is consistent with the previous literature having found effects of exposure to prenatal shocks on gender from conception up to month five (see Section 2.2).[19]

A similar analysis of the effect of conflict exposure on the probability of stillbirth (Table 5) shows that exposure to conflict *in utero* leads to a small decrease in the probability of stillbirth in absolute terms (0.22 ppt when controlling for maternal unobserved heterogeneity). Contrary to the findings for miscarriage, however, controlling for maternal fixed effects does not affect the findings for stillbirths, suggesting that the determinants of stillbirths (or their reporting) are less closely related to maternal unobserved heterogeneity than determinants of miscarriage. Intrapartum deaths make up 50% of stillbirths in Nepal (McClure et al. 2006*b*). Many of these deaths can be prevented by appropriate medical attention during delivery, and the quality of this medical attention may be well captured by district fixed effects. In addition, individual heterogeneity in reporting a stillbirth is likely

[19]Medical studies suggest that pregnancy outcomes can be affected by maternal psychological stress experienced up to month 6 of pregnancy (see: Glynn et al. (2001) for a reduction in gestational length increasing with exposure earlier in the pregnancy; Torche & Kleinhaus (2012) for an effect on preterm birth for exposure during months 2 and 3; Dancause et al. (2011) for an increase in preterm births and lower birth weight for exposure during early to mid-pregnancy; Class et al. (2011) for an effect until months 5 and 6, and especially during these months, on gestational age, preterm birth, low birth weight and size for gestational age. Evidence on the impact of maternal malnutrition on fetal and infant health tends to suggest that it is most detrimental the later it occurs in pregnancy (Stein & Susser (1975), Roseboom et al. (2001), and Painter et al. (2005). In a notable exception, Almond & Mazumder (2011) recently find that *in utero* exposure to maternal fasting during the holy month of ramadan decreases birth weight most when it occurs during the first two trimesters.

to be less than individual heterogeneity in reporting a miscarriage. Results in Column (4) indicate that the decrease in the probability of stillbirth is driven by exposure to conflict up to mid-gestation, which we have shown to cause an increase in the probability of miscarriage and decreased the sex-ratio. One plausible explanation for the observed decrease in the probability of stillbirth therefore is that conflict exposure leads to selection on fetal health through miscarriage.

The last column of Tables 3, 4 and 5 report estimates of the effect of exposure by trimester. Based on the existing evidence and the fact that most miscarriages occur by month five, predictions in terms of the effect of exposure by trimester on miscarriage, stillbirth and thus gender at birth are not clear-cut. While some studies have found effects on gender for exposure during the first trimester, others only found effects for exposure during the first half of the second trimester (e.g., Catalano et al. (2006)). One could therefore observe a significant increase in miscarriage and the probability of giving birth to a girl following first-trimester exposure, or not. In addition, exposure late into the second trimester should not affect miscarriages, and therefore second-trimester exposure may not be found to increase the probability of miscarriage or of a female birth even when exposure in the early second trimester has an effect. Finally, there should be no significant effect of third trimester exposure on miscarriage, but the existing evidence does not offer much guidance in terms of the expected effect on stillbirth and gender at birth for a developing country context, where intrapartum stillbirths are much more common and given that male infants are more at risk of stillbirth (McClure et al. 2007). Indeed, poor quality of medical care during delivery, which is an important risk factor for stillbirths in developing countries (McClure et al. 2007), may be reinforced by conflict intensity in the third trimester for a number of reasons including restricted travel and low availability of medical supplies or personnel. Third trimester conflict exposure may therefore increase the probability of stillbirth and thus increase the probability of a live born infant being female. I find that both first- and third-, but not second-trimester exposure significantly increase the probability of a female birth (Table 3). Similarly, in the case of miscarriage, the largest point estimate is found for first trimester exposure, but none of the trimester-specific exposure point estimates is statistically significant (Table 4). For stillbirth, only second-trimester exposure leads to a statistically significant decrease in the probability of stillbirth, although the point estimate for first trimester exposure is larger in magnitude. On the contrary, the sign of the coefficient for third trimester exposure is positive although insignificant, which could be due to conflict intensity close to delivery, but not throughout the third trimester, increasing the risk of

stillbirth.

Taken together, the findings for exposure by first and second half of pregnancy and for exposure by trimester suggest that exposure to conflict during the first five months of pregnancy matter for miscarriage and gender at birth, but that exposure in the late second trimester does not, and that this increased risk of miscarriage may lead to positive selection in terms of the probability of stillbirth. In addition, exposure in the third trimester also increases the probability of a female birth, which could follow from an increased chance of stillbirth due to conflict activity close to birth.

In the next section, I further probe the TW selection hypothesis and discuss the implications of my findings for the treatment of gender in applied econometric models.

6 Scarring or Selection? Findings and Implications for Applied Econometric Models

The results presented above indicate that gender is not exogenous in the sense that it is partly determined by transitory shocks during pregnancy. The precise implications of this finding for the interpretation of models commonly estimated in the applied economics literature depend on the mechanism at play (scarring or selection). In this section, I present my findings on the effect of exposure to conflict *in utero* on health at birth, and interpret these findings in the light of the analytical framework of Section 2.3, concluding that one mechanism does not appear to dominate another. I then discuss the implications of my findings for applied econometric models, which should encourage researchers to (i) explore the extent to which the effect of prenatal shocks on later life outcomes may be mediated by gender and (ii) take into account the fact that female gender may in part capture an omitted variable, be it parental socioeconomic and health status, or the degree of scarring or selection experienced by the individual's birth cohort.

6.1 Scarring or Selection: Findings

As explained in Section 2.3, an increase in miscarriages can account for a decline in the sex-ratio either through (i) a worsening of the distribution of fetal health, or (ii) an increase in the threshold required for fetal survival, or both. In both cases, more male fetuses are spontaneously aborted because they are over-represented at the lower end of the fetal health distribution. According to Catalano & Bruckner (2006), the Trivers-Willard hypothesis

is only verified under (ii). From the point of view of applied econometric models, whether (i) or (ii) dominates matters for the interpretation of results obtained when conditioning on gender, since under case (ii), female gender would tend to be correlated with belonging to more positively selected cohorts, whereas under case (i), it would be correlated with more scarred cohorts.

So far I have found that the probability of stillbirth significantly decreases due to maternal exposure to conflict during the first half of pregnancy, suggesting an increase in the threshold required for survival to late pregnancy. The increase in the probability of miscarriage is larger than the decrease in the probability of stillbirth, so that the total effect on the likelihood of survival to (live) birth is a decrease in the probability of a live birth, thus opening the possibility of positive selection into live birth.[20] In order to further test the selection hypothesis, I estimate the impact of exposure to conflict *in utero* on health at- and shortly after birth, as captured by small size at birth and neonatal mortality (both self-reported by the mother).

Results are reported in Table 6. Column (3) of Panel A shows that children whose mothers experience more intense conflict during pregnancy are less likely to be small compared to their siblings who were *in utero* at times of less intense conflict. A similar sign is obtained when comparing children within district rather than within sibship (Columns (1) and (2)), but the estimate is only statistically significant when maternal unobserved heterogeneity is controlled for.[21] Turning now to the first three columns of Panel B, we can see that exposure to conflict *in utero* is correlated with a lower probability of dying within the first month of life, but this correlation becomes insignificant when including maternal fixed effects.

In the two next columns, I consider separately exposure in the first- versus the second half of pregnancy (Column (4)) and in the three different trimesters (Column (5)). When splitting the pregnancy period in two halves, i.e. when splitting it by exposure that affects miscarriage (first half) and exposure that does not (second half), I find that exposure in the first half of pregnancy has a statistically significant, negative effect on the probability of a baby being small at (live) birth, while the effect on neonatal mortality is negative but statistically insignificant for both exposure vari-

[20] When estimating the effect of exposure to conflict *in utero* on a dummy variable equal to 1 if a pregnancy ends in either a miscarriage or a stillbirth, and 0 if it ends in a live birth, the total effect is a 0.68 ppt decrease in the probability of a live birth (within-mother), and is statistically significant at the 10 percent level. Results are available on request.

[21] I have experimented with a dependent variable taking a different value for each possible answer: 1 for "very small", 2 "smaller than average", 3 "average", 4 "larger than average" and 5 "very large". My conclusions are not sensitive to this change in specification. Results are available on request.

ables. In Column (5), I find that the negative effect of exposure to conflict on small size at birth and on neonatal mortality is statistically significant for both outcomes when experienced in the second trimester, suggesting that the selection mechanism is strongest for exposure to conflict in the early second trimester.

Taken together, the results for small size at birth and neonatal mortality give limited support for the selection hypothesis. In order to further probe this interpretation, I test another implication of the selection hypothesis, which is that the probability of poor health at birth should decrease more in absolute terms for newborn boys than girls, since the mean health endowment of the fetuses lost due to the shift in the survival threshold should be lower among males. Column (7) of Table 6 presents results from regressions of an indicator for small size at birth (Panel A) and for neonatal mortality (Panel B) obtained when estimating an augmented version of Equation 4 including a female indicator and interaction terms between this indicator and the conflict variables. The coefficients on the interaction between female gender and exposure to conflict *in utero* are not statistically significant, but are negative and large compared to the effect of exposure *in utero* on males, suggesting that the probability of poor health at birth does not decrease more for newborn boys than girls and thus failing to support the TW hypothesis as the sole cause for my findings. However, there is no indication that children exposed to more conflict in the first half of pregnancy and born alive are in worse health, thus suggesting that scarring alone cannot explain the increase in miscarriage and in female births, and that it must be the result of both scarring and selection mechanisms.

The results on size at birth contrasts with recent findings by Camacho (2008) for Colombia and Mansour & Rees (2012) for Palestine. Both Camacho (2008) and Mansour & Rees (2012) find that conflict events have a negative effect on birth weight when experienced during the first-trimester and, in some specifications, Mansour & Rees (2012) find a negative effect of exposure during the third trimester, but this is not the case here. In a regression similar to the within-mother specification in Panel A of Table 6 (Column (5)) where pre_{dt} and $inUtero_{dt}$ are replaced by three *in utero* conflict exposure corresponding to mean monthly exposure during the three trimesters defined as in Mansour & Rees (2012) ($mc+1$ to $mc+3$, $mc+4$ to $mc+6$, and $mc+7$ to $mc+9$), the effect of exposure to conflict during the first two trimesters on the probability of small size at birth is negative (i.e., more conflict is associated with larger size at birth), and statistically significant in the first and second trimester, while the effect of exposure during the third trimester is positive but statistically insignificant.[22]. Fur-

[22]Coefficients and standard errors are as follows. Trimester 1: -0.594 (0.2583);

ther research is needed in order to understand the circumstances under which scarring effects are more likely to dominate selection. One hypothesis based on findings from Colombia, Palestine, and Nepal is that selection may be stronger in less developed settings, where baseline undernutrition may compound the effect of psychological stress on fetal growth (Cliver et al. 1992).

6.2 Implications for Applied Econometric Models

What are the implications of these findings for applied econometric models? As pointed out by Almond & Edlund (2007), gender is not exogenous in the sense that it is partly determined by maternal condition, be it in terms of socio-economic status as in Almond & Edlund (2007), or, as shown here, in terms of transitory shocks during pregnancy for a given mother. Caution should therefore prevail when controlling for gender in empirical work, especially in the case of studies interested in the impact of shocks *in utero* on later outcomes, which are growing in popularity.

There are two types of issues to be considered. First, part of the causal relationship of interest may be mediated by the effect of shocks occurring during gestation on gender, as can be illustrated in the context of the present study. We have seen that conflict exposure *in utero* increases the probability of a female birth. It is also well-known that female babies are smaller than male babies, on average (National Center for Health Statistics 2001), and have an inherently lower risk of neonatal mortality (Naeye et al. 1971). Therefore, exposure to conflict may lower size at birth and decrease the risk of neonatal death through its effect on gender. Column (4) of Table 6 show estimates obtained when regressing indicators for small size at birth (Panel A) or neonatal mortality (Panel B) on the set of regressors included in Equation 4 and a control for child gender. As expected, the coefficient on conflict exposure *in utero* increases in magnitude in Panel A, and decreases in magnitude in Panel B. The part of the *in utero* shock mediated by its effect on gender may be particularly large for outcomes with very different prevalence or distributions among males and females, such as cardiovascular disease and income, which have been studied in tests of the fetal origin hypothesis. Second, female gender may in part capture an omitted variable, be it parental socioeconomic and health status, or the degree of scarring or selection experienced by the individual's birth cohort. In the case of parental SES or health status, the TW hypothesis, when verified, implies that a control for female gender would be correlated with lower parental status. In the case of a maternal stressor occurring during

trimester 2: -0.804 (0.2486); trimester 3: 0.451 (0.8098)

pregnancy, as in the present study, whether female gender is a marker for a more "scarred" cohort or a more "selected" cohort depends on whether the decrease in the sex-ratio is the result of a shift of the fetal health distribution to the left, or that of an increased survival threshold. In the former case, female gender would tend to be correlated with a poorer health endowment at birth, whereas in the latter case, female gender would be correlated with a better health endowment at birth.

In the case of armed conflict, the present analysis suggests that prenatal exposure increases the threshold fetuses have to exceed in order to survive to late pregnancy (since the probability of stillbirth decreases somewhat when the probability of miscarriage increases), but from birth onwards, it is not clear that the increase in the survival threshold dominates the leftward shift of the fetal health distribution. This suggests that both selection and scarring forces are at work and that only part of the decrease in the sex-ratio can be attributed to a TW mechanism. The robustness checks presented in the following section reinforce this conclusion.

7 Robustness Checks

In Table 7, I report a number of checks on the robustness of my main results based on the maternal fixed effects specification. As detailed in Section 4.2, in the main analysis I include a number of controls over and above the regressor of interest, which is maternal exposure to conflict *in utero*, in order to control for time-varying factors which may be correlated with both exposure to conflict during gestation and the outcome of interest. In Panel A, I check that my results hold when I exclude these controls, and they do. The only difference with the main set of results is that the effect of exposure to conflict *in utero* on small size at birth is not statistically significant, reinforcing the conclusion that for those babies being born alive, selection does not significantly dominate the scarring effect of exposure to conflict *in utero*.

Mother fixed effects control for average circumstances of the mother during the five year period covered by the data. It is, however, possible that fertility decisions are influenced by factors that vary within-mother over this five-year period. If these factors are uncorrelated with conflict, then this should not affect the results (for instance, crop failure uncorrelated with conflict). Some sources of selection may, however, be correlated with conflict intensity. If they are correlated with the cumulated intensity of conflict, then they are controlled for by pre_{dt} (for instance, a cumulative worsening of income earning opportunities). If they are correlated with recent events before conception, then they should be picked up by a placebo

treatment corresponding to conflict intensity during the year or two preceding pregnancy. The placebo tests results discussed below and reported in Tables 7 and A-1 show that this is not a concern except for the size at birth outcome.

In Panel B of Table 7, I include a placebo treatment variable corresponding to monthly average district conflict intensity during the same calendar period as the index pregnancy, but the year before the pregnancy took place. If conflict intensity during gestation is correlated with underlying trends in the explained variable, then the placebo treatment should pick up such trends. For all outcomes except small size at birth, the coefficient on the placebo treatment variable is statistically insignificant and much smaller in magnitude than the effect of exposure to conflict during pregnancy, which bolsters confidence in the identification strategy. In the case of small size at birth, it appears that the placebo treatment effect is very similar to the actual treatment effect, and statistically significant, further reinforcing the conclusion that there is no robust evidence here of selection dominating scarring conditional on survival to (live) birth. Panels A, B and C of Table A-1 present additional placebo tests, excluding pre_{dt} and varying the period of "placebo" exposure. The only noticeable difference with Panel B of Table 7 is that conflict during the two years preceding conception increase the probability of a neonatal death - this is in line with the correlation between cumulated conflict before conception and neonatal mortality noted in the main regression results.

Panel C of Table 7 provides an additional check of the robustness of my findings to underlying trends, as results presented in this panel are net of district-specific linear trends. The effects of conflict exposure on miscarriage and gender are similar, although somewhat larger in magnitude, while the effect of conflict on stillbirth and small size at birth become insignificant, thus echoing the conclusion that there is no robust evidence here of selection dominating scarring. Panel D of Table A-1 reports findings for the same regression except for the exclusion of pre_{dt}, and show similar effects on miscarriage and gender.

Controlling for mother age and pregnancy order also addresses some of the potential source of (within-mother) selection into becoming pregnant by addressing the issue that older women and women who have fewer children may be more likely to try to conceive despite high conflict intensity. All in all, although sources of time-varying selection into becoming pregnant cannot be completely ruled out, they appear unlikely to be a substantial source of bias in the present analysis.

In Panel D of Table 7, I test the robustness of my findings to migration patterns. Recall that, in the main sample, I drop pregnancies which took

place before mothers moved to the place where they are interviewed. Indeed, the survey did not collect full migration histories from interviewees, only the length of residence in their current domicile, and therefore I do not know in which district a pregnancy took place if it happened before the mother moved to the place where she is interviewed. Dropping pregnancies which cannot be accurately assigned a district conflict intensity reduces (classical) measurement error in exposure to conflict. One potential problem arising when dropping these observations is that the sample of women who have moved during the period covered may be a selected sample in ways that matter for the effect of conflict on pregnancy outcomes. Mother fixed-effects estimates would still be internally valid, but such selection would restrict the external validity of findings based on this sample, especially if migration was caused by conflict. However, conflict is unlikely to account for more than a negligible share of women who have migrated. In a regression of total district deaths per 1,000 inhabitants in the current district of residence on a dummy for having migrated, I obtain a coefficient of 0.015 (s.e.:0.0241) on migration status (for a sample mean of 0.78 and s.d. of 0.683 for the dependent variable). This indicates that mothers who have migrated have not, on average, moved to low-conflict areas. Furthermore, I refer to the more detailed migration data collected by the Living Standards and Measurement Surveys of 2003/04 and 2010/11, which collected migration information for each household member aged 5 or above. After being asked whether they had migrated to their current place of residence, respondents were asked the reason for their migration. In 2003/04, only 2 (8) out of 1,386 migrants reported "political reasons" ("escaping/natural disaster") as the reason for their migration (own calculations based on Central Bureau of Statistics [Nepal] (2004)).[23] I do not have access to the 2010/11 dataset, but the survey report lists the following breakdown of causes for migration: "marriage" (53%), "family reason" (27%), "easier life style" (7%), "education and training" (5%), "looking for job" (4%), and "others" (4%) (Central Bureau of Statistics [Nepal] (2011), p.132) - the questionnaire has a "conflict" answer code to this question, which is lumped with various other answers in the 4% "others" mentioned in the report. This seemingly rare incidence of conflict-induced migration is consistent with estimates of internally displaced people - about 200,000 people (i.e. less than 1 percent of the population) according to (US Agency for International Development 2007). All in all, conflict-induced migration has not been as pronounced a phenomenon in Nepal as in many other conflict episodes, so that the improvement in precision obtained by restricting

[23]The breakdown of other specified reasons was as follows: "family reasons" (1,142), "easier lifestyle" (98), "looking for work" (56), "others" (52) and "education/training" (28).

the sample to "non-movers" should not come at too high a cost in terms of migration bias. In order to ascertain this prior, in Panel D I include pregnancies which occurred before the respondent came to live in her current place of residence. I allocate to these pregnancies the degree of conflict intensity in their mother's current district of residence at the time of the pregnancy, although a number of these pregnancies will have taken place in an unknown different district. The key findings that miscarriages increase and the sex-ratio decreases hold, although the magnitude of the estimated effects decreases somewhat, as would be expected in the presence of classical measurement error.

Prenatal exposure to conflict may increase the occurrence of preterm birth. Therefore, assuming a 9-month gestational period for live births may introduce some measurement error. In Panel E of Table A-1, I test the robustness of my findings to assuming an 8-month gestational period instead, and find that results are essentially unchanged.

Finally, further robustness checks in Table A-2 test the robustness of my findings to excluding children of women who have been married more than once and to excluding conflict intensity outliers. The first check tests the robustness of my maternal fixed effects findings to a change in the identity of the father, since children of different fathers may not be comparable in terms of their probabilities of being female or healthy. The DHS only collects the date of the woman's first marriage, and therefore I cannot assign children to fathers. However, I can exclude all mothers who have been married more than once (and therefore drop siblings with different fathers as well as siblings with the same father). My conclusions are unchanged (see Panel A2). Excluding three outliers from the pregnancy sample,[24] results in Panel B2 show that conclusions are unchanged, despite a decrease in statistical significance.

8 Conclusion

Previous studies have found a decrease in the sex-ratio coinciding with *in utero* exposure to a number of maternal stressors, including earthquakes (Fukuda et al. (1998); Torche & Kleinhaus (2012)), terrorist attacks (Catalano et al. 2006), and maternal fasting (Almond & Mazumder 2011). The timing of the maternal stressor with respect to the stage of pregnancy has provided indirect evidence that this skewing is likely to be due at least in part to disproportionate loss of male fetuses post-fertilization rather than changes in

[24] For these three observations, the number of casualties during pregnancy is (factually correct but) much higher than the next highest value. More specifically, after nine observations with exposure between .19 and .22, the three outlying values are .27, .29 and .48.

the sex-ratio at conception. This paper provides direct evidence that civil conflict experienced in the first half of pregnancy both increases the probability of spontaneous abortion and decreases the ratio of male-to-female newborns, holding maternal unobserved heterogeneity constant.

Tests of the effect of maternal characteristics or environmental shocks on the sex-ratio at birth are often cast in the light of the TW hypothesis, but several mechanisms have been put forward in order to account for changes in sex-ratios, which could result in the observed changes in the sex-ratio in their own right. For instance, disproportionate male fetal loss could arise due to a worsening of fetal health across the board, which, given the more pronounced frailty of the male fetus compared to its female counterpart, would push more male than female fetuses below a set survival threshold. Under the TW hypothesis, however, a decrease in the sex-ratio resulting from a worsening of maternal condition after conception arises due to an evolved culling mechanism, i.e., an increase in the fetal health endowment required to survive to birth (Catalano & Bruckner 2006). One implication of this evolved shift in the fetal survival threshold is that surviving fetuses should be in better health, i.e., they should be positively selected. Here I find some evidence of selection into late pregnancy, in the sense that the probability of stillbirth decreases somewhat with exposure to conflict during the first half of pregnancy. However, conditional on live birth, there is no robust evidence that *in utero* exposure to conflict causes a significant change in the probability of being small at birth or of neonatal death, thus suggesting that both selection and scarring forces are at work and that only part of the decrease in the sex-ratio can be attributed to a TW mechanism.

From a policy point of view, this study stresses the need to consider public health policies aimed at supporting women in conflict situations to deal with the trauma of pregnancy loss, even where usual indicators of newborn health such as size at birth and neonatal mortality do not worsen. From the point of view of increasingly popular studies interested with the impact of shocks *in utero* on later outcomes, the present findings will encourage authors to routinely test for the presence of effects mediated by gender. Finally, from the point of view of any applied economic analysis controlling for gender, this study flags the potential for the gender covariate to capture omitted variables not only relating to parental characteristics, as suggested before (Almond & Edlund 2007), but also relating to an individual's *in utero* experiences.

Table 1: Summary Statistics

District Conflict-Intensity Tercile	(1) Low			(2) Intermediate			(3) High		
	mean	sd	count	mean	sd	count	mean	sd	count
Live births sample									
Explained variables									
=1 if female[c]	0.508		3892	0.482		3403	0.498		3296
=1 if neonatal death	0.035		3892	0.035		3403	0.035		3296
=1 if baby smaller than average[a],[c]	0.165		3892	0.242		3403	0.239		3296
Pregnancy characteristics									
=1 if first pregnancy	0.226		3892	0.210		3403	0.213		3296
=1 if second pregnancy	0.229		3892	0.216		3403	0.227		3296
=1 if third pregnancy[a],[c]	0.193		3892	0.169		3403	0.163		3296
=1 if fourth pregnancy	0.125		3892	0.127		3403	0.127		3296
=1 if fifth pregnancy and higher[a],[c]	0.226		3892	0.278		3403	0.270		3296
Maternal age at conception[a],[b],[c]	24.106	5.9514	3892	24.885	6.3959	3403	24.485	6.3194	3296

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District Conflict-Intensity Tercile	(1) Low			(2) Intermediate			(3) High		
	mean	sd	count	mean	sd	count	mean	sd	count
Maternal characteristics									
=1 if urban[a],[b],[c]	0.186		3892	0.097		3403	0.119		3296
=1 if no education[a],[b],[c]	0.663		3892	0.716		3403	0.755		3296
=1 if primary education	0.149		3892	0.154		3403	0.140		3296
=1 if secondary education[a],[b],[c]	0.165		3892	0.116		3403	0.097		3296
=1 if higher education[a],[b],[c]	0.023		3892	0.014		3403	0.007		3296
=1 if Dalit[b],[c]	0.170		3892	0.140		3403	0.165		3296
=1 if Brahmin or Chhetri[a],[b],[c]	0.214		3892	0.379		3403	0.423		3296
=1 if Tarai/Madhesi Others[a],[b],[c]	0.256		3892	0.046		3403	0.013		3296
=1 if Newar	0.034		3892	0.032		3403	0.037		3296
=1 if Janajati[a],[b],[c]	0.195		3892	0.395		3403	0.334		3296
=1 if Muslim[a],[b],[c]	0.107		3892	0.005		3403	0.024		3296
=1 if Other Caste[a],[b],[c]	0.025		3892	0.001		3403	0.005		3296
Conflict exposure - district casualties per 1000 inhabitants									
Before conception, total[a],[b],[c]	0.039	0.0783	3892	0.118	0.2124	3403	0.423	0.7514	3296
During pregnancy, monthly average[a],[b],[c]	0.002	0.0033	3892	0.005	0.0086	3403	0.011	0.0207	3296

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District Conflict-Intensity Tercile	(1) Low			(2) Intermediate			(3) High		
	mean	sd	count	mean	sd	count	mean	sd	count
Pregnancy sample									
Explained variables									
=1 if miscarriage	0.054		4226	0.048		3652	0.054		3564
=1 if stillbirth[b],[c]	0.013		4047	0.009		3507	0.014		3422

Mean values for each explained variable are reported at the bottom of the corresponding results tables. The Nepal DHS contains 96 ethnicity categories. Here they are grouped following Bennett et al. (2008). The miscarriage and stillbirth indicators are equal to zero for live births, and set to missing if the mother does not answer "No" when asked if any action was taken to end the pregnancy. Source: Author's calculations using Nepal DHS 2001 and DHS 2006, Informal Sector Service Center (2009) and Central Bureau of Statistics [Nepal] (2009). [a] Difference between "Low" and "High" categories is statistically significant at the 10% level. [b] Difference between "Intermediate" and "High" categories is statistically significant at the 10% level. [c] Difference between "Low" and "Intermediate" categories is statistically significant at the 10% level.

Table 2: Selection on Observable Maternal Characteristics

	Asset Ownership Quintile				
	(1)	(2)	(3)	(4)	(5)
	First (Lowest)	Second	Third	Fourth	Fifth
Before	0.060	-0.038	0.011	-0.031	-0.003
conception	(0.0438)	(0.0372)	(0.0270)	(0.0270)	(0.0157)
During	-0.374	0.348	0.221	0.046	-0.241
pregnancy	(0.3868)	(0.3752)	(0.3589)	(0.3339)	(0.1996)
Mean Y_{imdt}	0.283	0.216	0.186	0.185	0.131
	Maternal Education				
	=1 if None	=1 if 1ary	=1 if 2ary	=1 if higher	=1 if Urban
Before	0.029	0.000	-0.013	-0.016**	0.003
conception	(0.0221)	(0.0160)	(0.0186)	(0.0062)	(0.0334)
During	0.091	0.158	-0.116	-0.133	-0.015
pregnancy	(0.2163)	(0.1785)	(0.1738)	(0.0843)	(0.3027)
Mean Y_{imdt}	0.703	0.149	0.131	0.016	0.141

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	Caste/Ethnicity						
	(1) =1 if Brahmin/ Chhetri	(2) =1 if Tarai/ Madhesi Others	(3) =1 if Dalit	(4) =1 if Newar	(5) =1 if Janajati	(6) =1 if Muslim	(7) =1 if Other Caste
Before conception	-0.093**	0.025	0.009	0.006	0.045	0.011	-0.005
	(0.0401)	(0.0152)	(0.0376)	(0.0089)	(0.0332)	(0.0214)	(0.0058)
During pregnancy	-0.830**	0.257	0.113	0.222	0.230	0.061	-0.054
	(0.3340)	(0.1646)	(0.4040)	(0.1448)	(0.3356)	(0.2251)	(0.0577)
Mean Y_{imdt}	0.340	0.111	0.160	0.034	0.296	0.048	0.011
Observations	11887	11887	11887	11887	11887	11887	11887

All regressions are estimated using the linear (district) fixed-effect estimator and include a constant and the following pregnancy characteristics: year of conception fixed effects, calendar month of conception dummies, age of mother at conception and its square, and 4 pregnancy order indicators. Binary indicators for wealth quintiles are provided in the DHS based on a principal component analysis of (i) ownership of consumer items such as television, bicycle, car, (ii) dwelling characteristics including source of drinking water, sanitation and type of housing materials. District-correlated robust standard errors in parentheses. District casualties are expressed per 1000 inhabitants. Casualties occurring during pregnancy are in monthly averages, whereas casualties occurring before conception are the cumulated number of casualties until the month before conception. Source: Author's calculations using Nepal DHS 2001 and DHS 2006, Informal Sector Service Center (2009) and Central Bureau of Statistics [Nepal] (2009). * p<0.10, ** p<0.05, *** p<0.01

Table 3: Effect of Conflict on the Probability of a Female Birth

	=1 if Female, =0 if Male				
	(1)	(2)	(3)	(4)	(5)
Before conception	-0.006	-0.008	0.050	0.049	0.036
	(0.0176)	(0.0208)	(0.0512)	(0.0515)	(0.0480)
During pregnancy	-0.330	-0.144	1.964*		
	(0.5019)	(0.6183)	(0.9949)		
Conception to conception+4				1.239**	
				(0.5049)	
Conception+5 to conception+9				0.723	
				(0.7872)	
1st trimester					1.027*
					(0.5333)
2nd trimester					-0.390
					(0.3322)
3rd trimester					1.007*
					(0.5947)
Year FE	Yes	Yes	Yes	Yes	Yes
Month FE	Yes	Yes	Yes	Yes	Yes
Pregnancy vars	Yes	Yes	Yes	Yes	Yes
Maternal vars	Yes	Yes	No	No	No
Observations	10591	5620	5620	5620	5620
No. of Groups	75	74	2674	2674	2674
No. of clusters	75	74	74	74	74
R-squared	0.0048	0.0064	0.0103	0.0104	0.0105
Mean Y	0.496	0.516	0.516	0.516	0.516

All regressions are estimated using the linear fixed-effect estimator. They all include a constant and the following pregnancy characteristics: year of conception fixed effects, calendar month of conception dummies, age of mother at conception and its square, and 4 pregnancy order indicators. Maternal characteristics ("vars"): a binary indicator for urban households, 3 maternal education dummies, and 6 caste/ethnicity indicators. District-correlated robust standard errors in parentheses. District casualties are expressed per 1000 inhabitants. Casualties occurring during pregnancy are in monthly averages, whereas casualties occurring before conception are the cumulated number of casualties until the month before conception. Source: Author's calculations using Nepal DHS 2001 and DHS 2006, Informal Sector Service Center (2009) and Central Bureau of Statistics [Nepal] (2009). * p<0.10, ** p<0.05, *** p<0.01.

Table 4: Effect of Conflict on the Probability of Miscarriage

	=1 if Miscarriage, =0 if Live Birth				
	(1)	(2)	(3)	(4)	(5)
Before conception	0.001	0.006	0.030	0.026	0.026
	(0.0086)	(0.0139)	(0.0236)	(0.0247)	(0.0239)
During pregnancy	0.203	0.465	0.854**		
	(0.2908)	(0.3790)	(0.3742)		
Conception to conception+4				0.705**	
				(0.2943)	
Conception+5 to conception+9				0.070	
				(0.4212)	
1st trimester					0.548
					(0.3640)
2nd trimester					-0.214
					(0.3094)
3rd trimester					0.443
					(0.3449)
Year FE	Yes	Yes	Yes	Yes	Yes
Month FE	Yes	Yes	Yes	Yes	Yes
Pregnancy vars	Yes	Yes	Yes	Yes	Yes
Maternal vars	Yes	Yes	No	No	No
Observations	11442	6594	6594	6594	6594
No. of Groups	75	74	3060	3060	3060
No. of clusters	75	74	74	74	74
R-squared	0.0083	0.0126	0.0487	0.0487	0.0487
Mean Y	0.052	0.066	0.066	0.066	0.066

Notes as under Table 3. See Section 4.1 for a detailed definition of miscarriage.

Table 5: Effect of Conflict on the Probability of Stillbirth

	=1 if Stillbirth, =0 if Live Birth				
	(1)	(2)	(3)	(4)	(5)
Before conception	-0.007	-0.013*	-0.007	-0.006	-0.005
	(0.0065)	(0.0077)	(0.0081)	(0.0081)	(0.0075)
During pregnancy	-0.106	-0.221**	-0.249*		
	(0.0872)	(0.1039)	(0.1329)		
Conception to				-0.234*	
conception+4				(0.1223)	
Conception+5 to				-0.001	
conception+9				(0.0530)	
1st trimester					-0.157
					(0.0974)
2nd trimester					-0.112**
					(0.0489)
3rd trimester					0.057
					(0.0494)
Year FE	Yes	Yes	Yes	Yes	Yes
Month FE	Yes	Yes	Yes	Yes	Yes
Pregnancy vars	Yes	Yes	Yes	Yes	Yes
Maternal vars	Yes	Yes	No	No	No
Observations	10976	6066	6066	6066	6066
No. of Groups	75	74	2866	2866	2866
No. of clusters	75	74	74	74	74
R-squared	0.0091	0.0148	0.0155	0.0158	0.0158
Mean Y	0.012	0.015	0.015	0.015	0.015

Notes as under Table 3. See Section 4.1 for a detailed definition of stillbirth.

Table 6: Effect of Conflict on Newborn Health

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Panel A				=1 if Small at Birth			
Before conception	-0.016	-0.022	-0.019	-0.018	-0.012	-0.022	-0.032
	(0.0254)	(0.0317)	(0.0304)	(0.0304)	(0.0292)	(0.0316)	(0.0353)
During pregnancy	-0.227	-0.859	-1.476*			-1.613*	-1.090
	(0.5537)	(0.5581)	(0.8827)			(0.8990)	(1.2390)
Conception to				-0.933***			
conception+4				(0.3510)			
Conception+5 to				-0.541			
conception+9				(0.7865)			
1st trimester					-0.409		
					(0.4024)		
2nd trimester					-1.207**		
					(0.4612)		
3rd trimester					0.501		
					(0.9075)		
=1 if Female						0.069***	0.072***
						(0.0123)	(0.0141)
Before conception							0.016
x =1 if Female							(0.0229)
During pregnancy							-0.985
x =1 if Female							(1.3566)
Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Month FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Pregnancy vars	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Maternal vars	Yes	Yes	No	No	No	No	No
Observations	10591	5620	5620	5620	5620	5620	5620
No. of Groups	75	74	2674	2674	2674	2674	2674
No. of clusters	75	74	74	74	74	74	74
R-squared	0.0147	0.0234	0.0217	0.0218	0.0232	0.0298	0.0300
Mean Y	0.213	0.219	0.219	0.219	0.219	0.219	0.219

(Continued)

Panel B	=1 if Neonatal Death						
Before conception	0.007	0.015	0.046**	0.046**	0.041**	0.047**	0.035
	(0.0094)	(0.0142)	(0.0191)	(0.0190)	(0.0199)	(0.0190)	(0.0224)
During pregnancy	-0.149	-0.327*	-0.272			-0.238	0.194
	(0.1341)	(0.1903)	(0.4475)			(0.4497)	(0.8105)
Conception to				-0.147			
conception+4				(0.2728)			
Conception+5 to				-0.126			
conception+9				(0.3660)			
1st trimester					-0.052		
					(0.2903)		
2nd trimester					-0.259*		
					(0.1318)		
3rd trimester					-0.122		
					(0.3253)		
=1 if Female						-0.018**	-0.017*
						(0.0075)	(0.0089)
Before conception							0.023
x =1 if Female							(0.0261)
During pregnancy							-0.855
x =1 if Female							(0.9741)
Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Month FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Pregnancy vars	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Maternal vars	Yes	Yes	No	No	No	No	No
Observations	10591	5620	5620	5620	5620	5620	5620
No. of Groups	75	74	2674	2674	2674	2674	2674
No. of clusters	75	74	74	74	74	74	74
R-squared	0.0094	0.0201	0.0319	0.0319	0.0323	0.0336	0.0342
Mean Y	0.035	0.049	0.049	0.049	0.049	0.049	0.049

Notes as under Table 3. See Section 4.1 for detailed definitions.

Table 7: Robustness Checks

	(1) Miscarriage	(2) Stillbirth	(3) Female	(4) Small Baby	(5) Neonatal
Panel A: No controls					
During pregnancy	0.696**	-0.164*	1.626**	-1.243	-0.640
	(0.3253)	(0.0889)	(0.7574)	(0.7577)	(0.4300)
Year FE	Yes	Yes	Yes	Yes	Yes
R-squared	0.0019	0.0044	0.0069	0.0067	0.0093
Panel B: Placebo experiments					
Before conception	0.033	-0.005	0.064	0.011	0.044**
	(0.0278)	(0.0076)	(0.0580)	(0.0333)	(0.0205)
During pregnancy	0.867**	-0.236*	2.062**	-1.276	-0.287
	(0.3621)	(0.1287)	(1.0296)	(0.8533)	(0.4426)
During pregnancy	-0.134	-0.087	-0.674	-1.372***	0.100
- 12 months[a]	(0.6593)	(0.0618)	(0.8279)	(0.4766)	(0.2879)
Year FE	Yes	Yes	Yes	Yes	Yes
Month FE	Yes	Yes	Yes	Yes	Yes
Pregnancy vars	Yes	Yes	Yes	Yes	Yes
R-squared	0.0487	0.0155	0.0105	0.0230	0.0320
Panel C: Controlling for district-specific linear trend					
Before conception	0.030	-0.004	0.107	-0.047	0.044*
	(0.0365)	(0.0103)	(0.0798)	(0.0599)	(0.0262)
During pregnancy	0.911**	-0.139	2.469**	-1.489	-0.449
	(0.4001)	(0.0962)	(1.0338)	(1.2046)	(0.5038)
Year FE	Yes	Yes	Yes	Yes	Yes
Month FE	Yes	Yes	Yes	Yes	Yes
Pregnancy vars	Yes	Yes	Yes	Yes	Yes
District Trends	Yes	Yes	Yes	Yes	Yes
R-squared	0.0638	0.0312	0.0288	0.0454	0.0459
Sample details for Panels A, B, and C (Baseline Sample)					
Observations	6594	6066	5620	5620	5620
No. of Groups	3060	2866	2674	2674	2674
Mean Y	0.066	0.015	0.516	0.219	0.049
Panel D: Sample including pregnancies which took place before migration to current place of residence (exposure measured with error)					
Before conception	0.022	-0.005	0.072	-0.048	0.036**
	(0.0239)	(0.0075)	(0.0466)	(0.0323)	(0.0166)
During pregnancy	0.792*	-0.062	1.604*	-1.577*	-0.180
	(0.4130)	(0.1658)	(0.8411)	(0.8531)	(0.4623)

(Continued on next page)

(Continued)					
Year FE	Yes	Yes	Yes	Yes	Yes
Month FE	Yes	Yes	Yes	Yes	Yes
Pregnancy vars	Yes	Yes	Yes	Yes	Yes
Observations	7393	6795	6290	6290	6290
No. of Groups	3421	3208	2991	2991	2991
No. of clusters	74	74	74	74	74
R-squared	0.0555	0.0119	0.0091	0.0254	0.0330
Mean Y	0.068	0.016	0.519	0.221	0.050

See notes under Table 3.[a]Average monthly number of casualties during the same calendar period as the gestation period, but 12 months earlier. For instance, if the pregnancy took place between January and September 2001, "During pregnancy - 12 months" refers to monthly average deaths during January and September 2000. The "before conception" is, as before, the cumulative measure of all conflict deaths up to the month prior to conception.

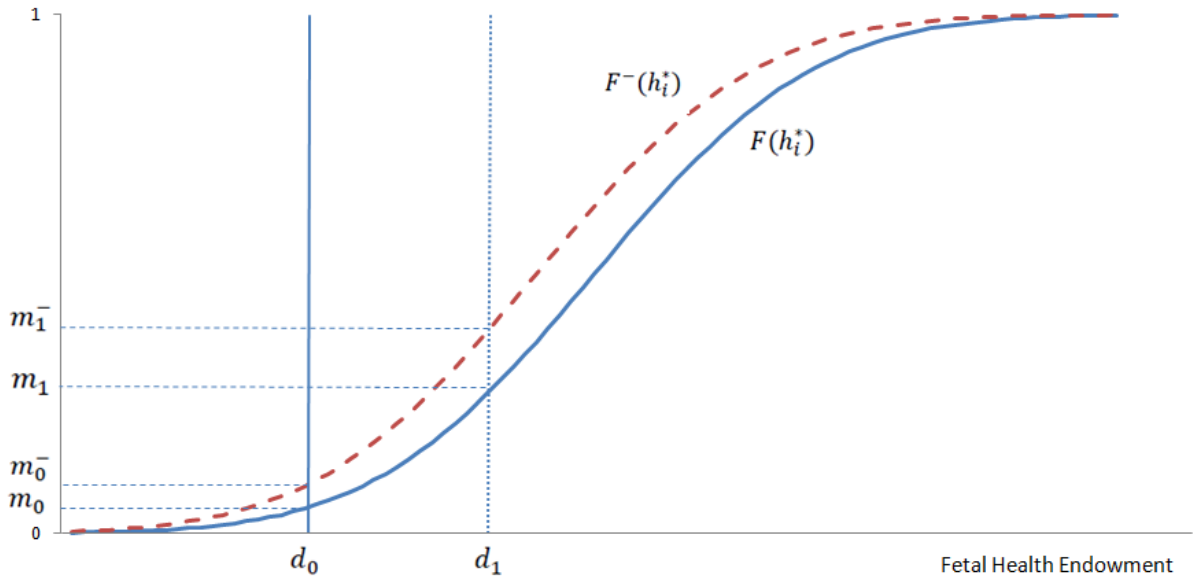

Figure 1: Scarring and Selection

h_i^* denotes the unobserved health endowment of individual i at conception, which, in an ideal health environment, is distributed according to the cumulative distribution $F(h_i^*)$. d_0 denotes the threshold below which fetuses cannot survive to birth, and m_0 is the fetal death rate. A negative health shock can either shift the fetal health distribution to the left to, e.g., $F^{-}(h_i^*)$ (scarring), or increase the survival threshold, to, e.g., d_1 (selection), or both.

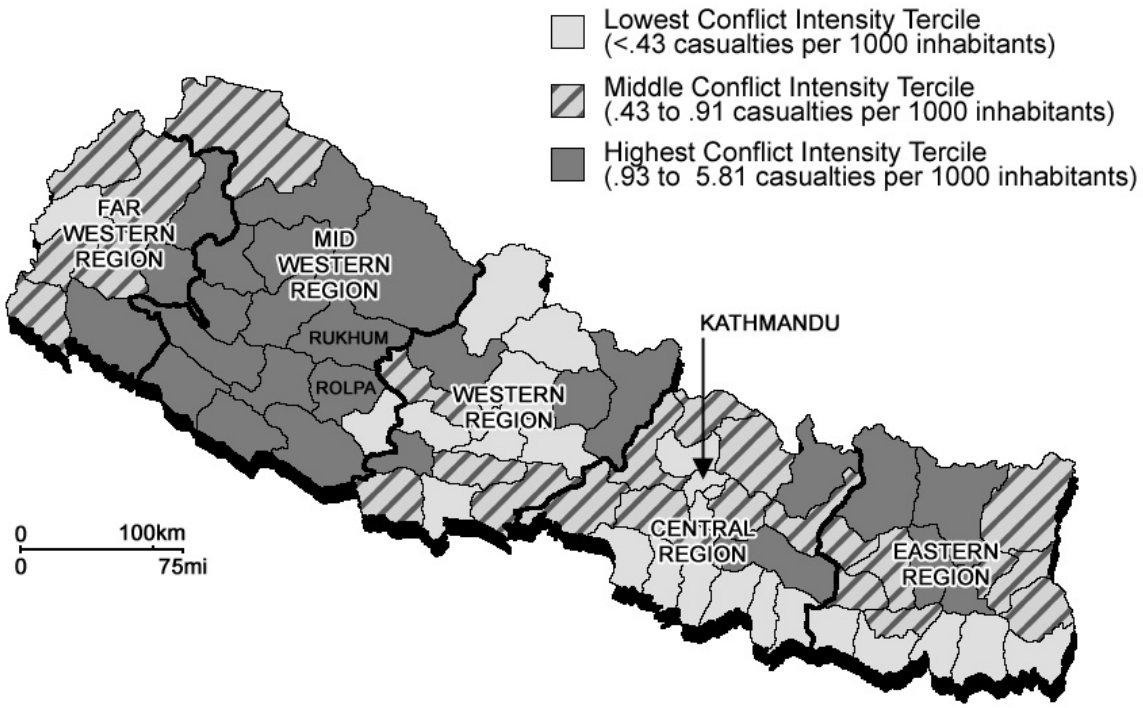

Figure 2: Between-District Variation in Casualties

Source: Author's calculation using data from Informal Sector Service Center (2009).

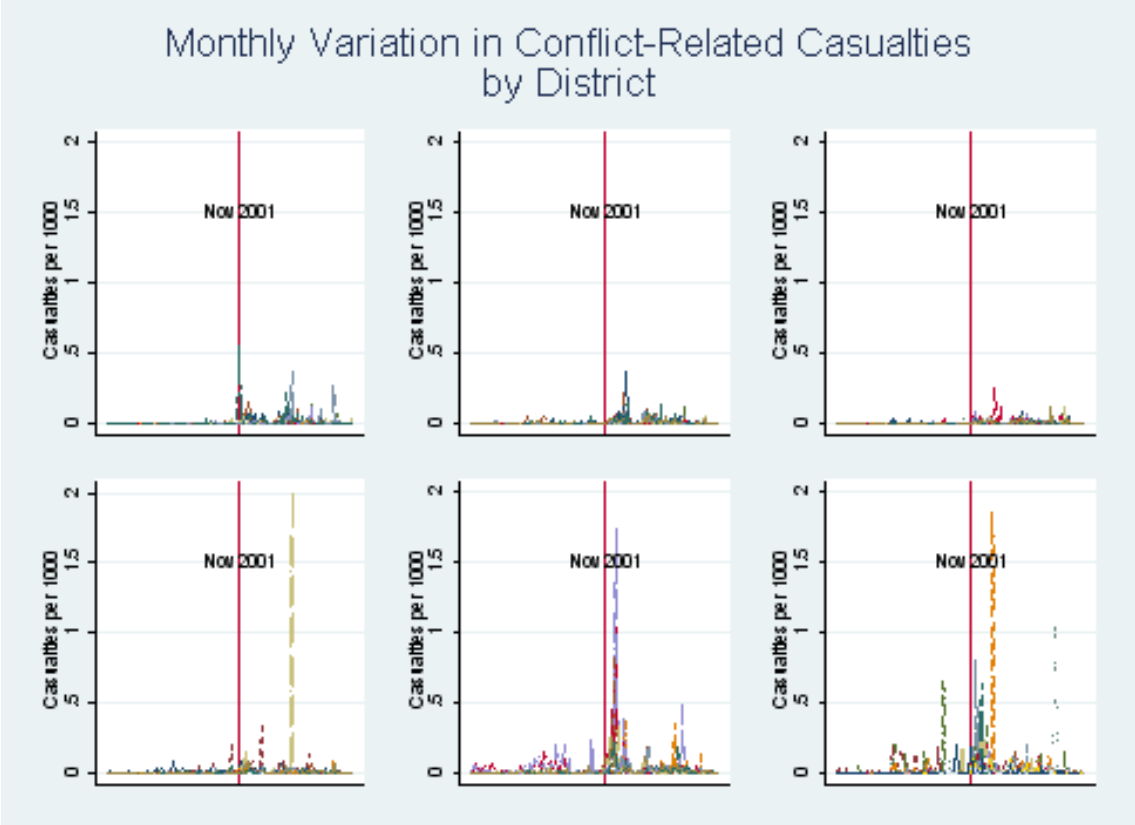

Figure 3: Within-District Variation in Casualties

The x-axis is a time line running from January 1996 to December 2006. November 2001 marks the start of conflict escalation. The 75 Nepalese districts are split into 6 groups and represented in separate diagrams, so that each line in each diagram plots monthly casualties for one of the 75 Nepalese districts over the course of the entire conflict.
Source: Author's calculation using data from Informal Sector Service Center (2009).

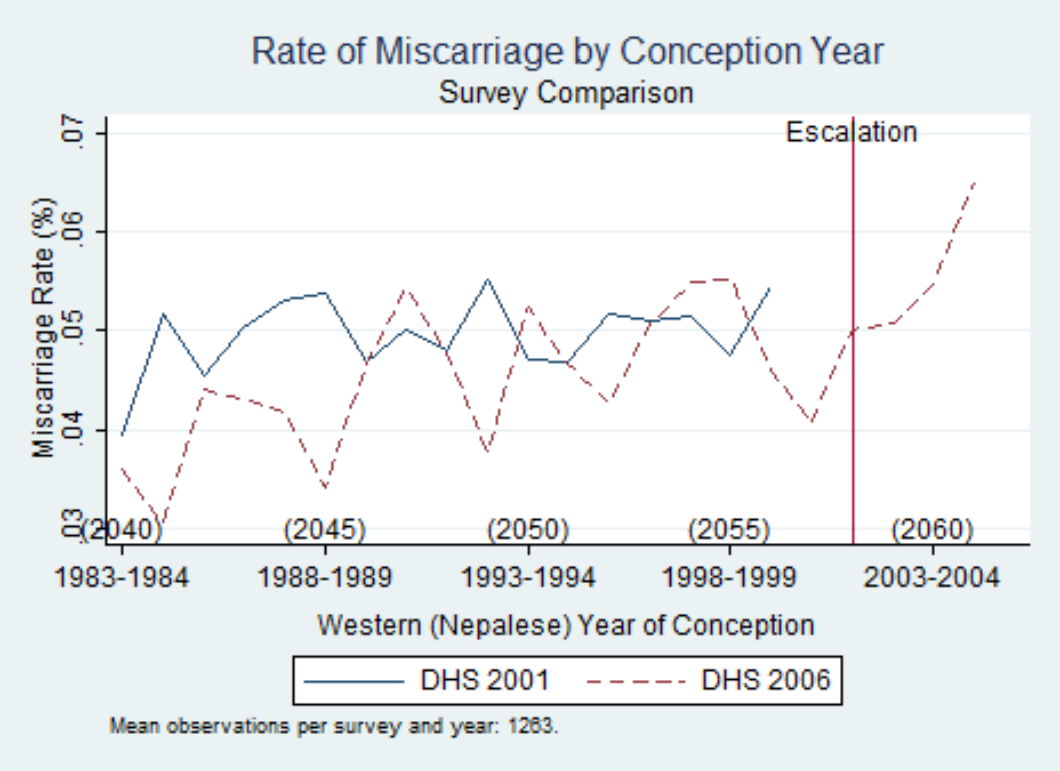

Figure 4: Reported Miscarriage Rates by Year and Survey Round

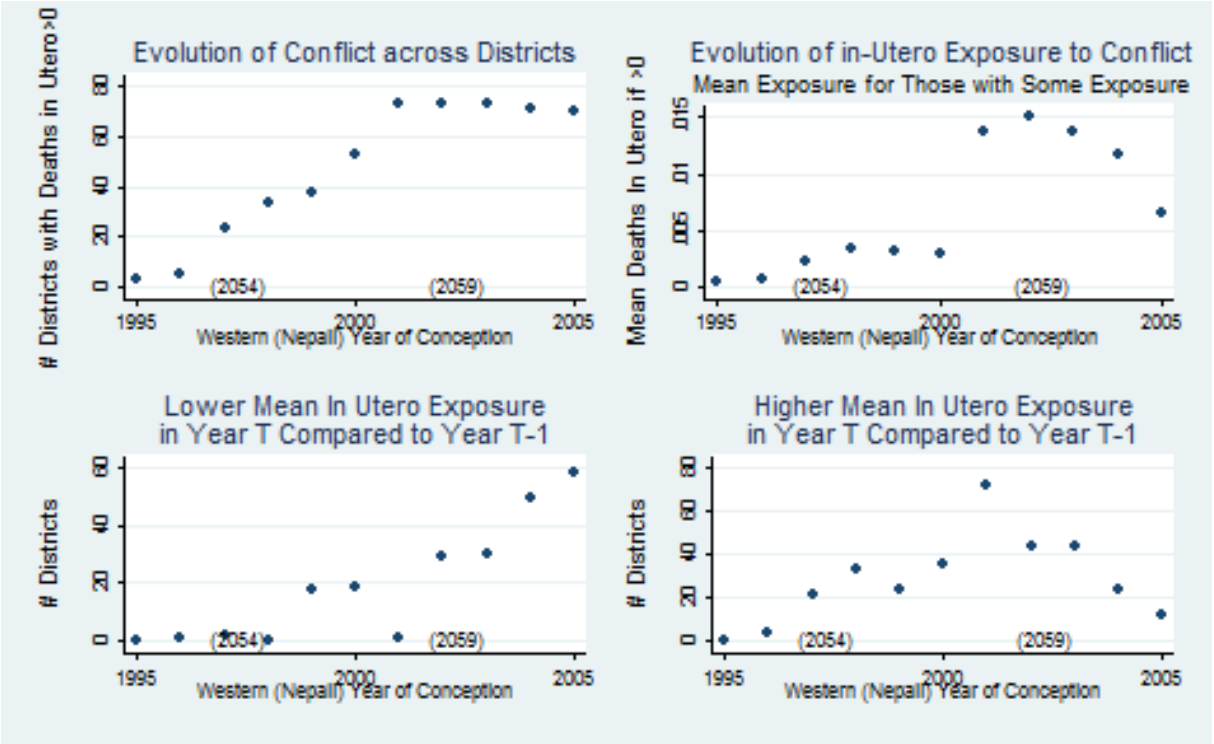

Figure 5: Variation in Conflict Intensity

Conflict deaths are expressed per 1,000 inhabitants.
Source: Author's calculation using data from Informal Sector Service Center (2009) and Central Bureau of Statistics [Nepal] (2009).

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Appendix

Table A-1: Further Robustness Checks I

	(1) Miscarriage	(2) Stillbirth	(3) Female	(4) Small Baby	(5) Neonatal
Panel A: Placebo test (pregnancy period lagged 12 months, excluding pre_{dt})					
During pregnancy	0.678**	-0.197**	1.528**	-1.370*	-0.649
	(0.3372)	(0.0898)	(0.7372)	(0.7765)	(0.4499)
During pregnancy - 12 months[a]	-0.005	-0.110	-0.358	-1.317***	0.315
	(0.6045)	(0.0833)	(0.7250)	(0.4234)	(0.2866)
R-squared	0.0483	0.0155	0.0102	0.0230	0.0310
Panel B: Placebo test (12 months before pregnancy, excluding pre_{dt})					
During pregnancy	0.678**	-0.194**	1.535**	-1.343*	-0.659
	(0.3268)	(0.0882)	(0.7352)	(0.7895)	(0.4475)
Pregnancy -12 to	0.239	-0.102	-0.585	-1.837***	0.180
Pregnancy -1[b]	(0.4296)	(0.0970)	(0.7460)	(0.4831)	(0.3223)
R-squared	0.0484	0.0155	0.0102	0.0238	0.0308
Panel C: Placebo test (24 months before pregnancy, excluding pre_{dt})					
During pregnancy	0.759**	-0.238**	1.620**	-1.489	-0.443
	(0.3641)	(0.1059)	(0.7881)	(0.9219)	(0.4024)
Pregnancy -24 to	0.262	-0.108	0.175	-0.392	0.499**
Pregnancy -1	(0.3042)	(0.0795)	(0.4400)	(0.5332)	(0.2127)
R-squared	0.0485	0.0155	0.0101	0.0218	0.0318

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Panel D: Controlling for district-specific linear trends					
and excluding the control for cumulated conflict exposure before conception					
During pregnancy	0.767**	-0.111	1.698*	-1.149	-0.766
	(0.3253)	(0.0983)	(0.9171)	(0.9740)	(0.5001)
R-squared	0.0636	0.0311	0.0282	0.0452	0.0454
Panel E: Assuming that live born children have an 8-month gestation period					
Before conception	0.028	-0.006	0.051	-0.022	0.045**
	(0.0237)	(0.0080)	(0.0522)	(0.0298)	(0.0189)
During Pregnancy	0.740**	-0.210*	1.923**	-1.513*	-0.291
(8 months if live)	(0.3640)	(0.1195)	(0.9540)	(0.8311)	(0.4295)
R-squared	0.0484	0.0154	0.0105	0.0219	0.0320
Sample details for Panels A-E (Baseline Sample)					
Observations	6594	6066	5620	5620	5620
No. of Groups	3060	2866	2674	2674	2674
Mean Y	0.066	0.015	0.516	0.219	0.049

See notes under Table 3.[a] Average monthly number of casualties during the same calendar period as the gestation period, but 12 months earlier, as in Table 7. For instance, if the pregnancy took place between January and September 2001, "During pregnancy - 12 months" refers to monthly average deaths during January and September 2000. [b] For instance, if the pregnancy took place between January and September 2001, "Pregnancy-12 to Pregnancy-1" refers to monthly average deaths between January and December 2000.

Table A-2: Further Robustness Checks II

	(1) Miscarriage	(2) Stillbirth	(3) Female	(4) Small Baby	(5) Neonatal
Panel A1: Women married only once					
Before conception	0.035	-0.008	0.036	-0.032	0.050**
	(0.0229)	(0.0088)	(0.0506)	(0.0272)	(0.0202)
During pregnancy	0.727*	-0.281*	1.578	-1.711**	-0.224
	(0.3922)	(0.1480)	(0.9744)	(0.7768)	(0.5076)
R-squared	0.0476	0.0138	0.0111	0.0216	0.0347
Panel A2: Women married only once, exposure in first vs. second half of pregnancy					
Before conception	0.028	-0.008	0.035	-0.031	0.050**
	(0.0243)	(0.0088)	(0.0509)	(0.0270)	(0.0201)
Conception to conception+4	0.579*	-0.247*	1.058**	-0.907***	-0.104
	(0.3095)	(0.1299)	(0.5120)	(0.3352)	(0.3009)
Conception+5 to conception+9	-0.087	-0.015	0.509	-0.802	-0.121
	(0.3676)	(0.0582)	(0.7699)	(0.7310)	(0.4034)
R-squared	0.0476	0.0141	0.0112	0.0216	0.0347
Sample details for Panels A1 and A2 (Sample of women married once only)					
Observations	6139	5664	5242	5242	5242
No. of Groups	2855	2681	2498	2498	2498
Mean Y	0.064	0.015	0.516	0.217	0.050
Panel B1: Exclude conflict intensity outliers					
Before conception	0.017	-0.006	0.051	-0.019	0.044**
	(0.0256)	(0.0080)	(0.0502)	(0.0299)	(0.0190)

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During pregnancy	0.301	-0.267*	1.677	-1.405	-0.010
	(0.6452)	(0.1434)	(1.0456)	(1.0256)	(0.4330)
R-squared	0.0474	0.0155	0.0096	0.0214	0.0316
Panel B2: Exclude outliers, exposure in first vs. second half of pregnancy					
Before conception	0.022	-0.005	0.051	-0.018	0.044**
	(0.0254)	(0.0077)	(0.0511)	(0.0301)	(0.0190)
Conception to conception+4	0.530*	-0.278**	1.018*	-0.911**	0.113
	(0.3149)	(0.1364)	(0.6001)	(0.4376)	(0.2001)
Conception+5 to conception+9	0.096	-0.006	0.698	-0.539	-0.097
	(0.4180)	(0.0541)	(0.7840)	(0.7920)	(0.3666)
Sample details for Panel B1 and B2 (Sample excluding outliers)					
Observations	6592	6065	5619	5619	5619
No. of Groups	3060	2866	2674	2674	2674
Mean Y	0.066	0.015	0.516	0.219	0.049

See notes under Table 3. Conflict-intensity outliers are three observations for which the number of casualties during pregnancy is (factually correct but) much higher than the next highest value. More specifically, after nine observations with exposure between .19 and .22, the three outlying values are .27, .29 and .48.